

St. Bartholomew's Hospital



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NOTICE.

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St. Bartholomew's Hospital Journal,

FEBRUARY 14th, 1896.

"Æquam memento rebus in arduis
Servare mentem."—Horace, Book ii, Ode iii.

IN response to the many requests from old Bart.'s men that we should establish a Pathological Department in connection with the JOURNAL, we have at last completed the necessary arrangements and now formally declare the Department open.

The question of a Pathological Department was far from being so simple as it appeared at first sight, and adverse criticisms on the project were not wanting from those who make it their practice to "let well alone," and who consider the sentence, "We've done very well without it up to the present" a sufficient answer to any suggestion of a new departure.

From the outset it was decided that the enterprise should not take the form of a financial speculation, and therefore that the charges should be just sufficient to cover expenses.

Men had frequently complained that in the absence of

a fixed charge they could not make use of the Pathological Department of the Hospital, much as they wanted to, and on the other hand, complaints came from the Pathological Laboratory that specimens were continually sent to them for report by old Bart.'s men who had never done anything for the Laboratory and whose names even were not familiar to the men who did the work.

The "Pathological Department of the Journal" is intended to meet both these difficulties. Specimens sent by subscribers of the JOURNAL will be examined and reported upon, at a charge which makes it just worth the while of the men engaged in the Laboratory to do the work. The amount paid for the report goes straight to the man who actually does the work, and neither the JOURNAL nor the Laboratory make any profit from the transaction.

Dr. Kanthack has most kindly promised to superintend the work and ensure the accuracy of the reports, but it must be understood that he is not in any way officially responsible for the working of the scheme. Needless to say, he has no connection with the financial arrangements.

The Department is open to anyone whose name is on the JOURNAL List, but *not* to non-subscribers. There is no intention of rivalling kindred organisations who have for their aim commercial advantage, but it is intended solely to aid old Bart.'s men in practice, whose interest in their cases makes them anxious for pathological reports, even though they hold out no prospect of advantage to the patient.

It need hardly be said that the practitioner is not expected to make any profit out of the examination, or to use the Pathological Department for the purpose of a cheap consultation, and when the examination is made with a view to the patient's advantage, and a fee is charged, it will of course be considered a point of honour that the whole of the fee paid for the work shall be forwarded to the Pathological Department.

The fees charged will be as follows:—For reporting upon a tissue, and making an ordinary bacteriological examination, such as that of sputum or membrane—half-a-crown. Where a telegraphic report is required the extra cost of telegraphing will be charged.

An ordinary report upon a urine—qualitative—will be charged half-a-crown, but any further examination will be made if required and a special charge in proportion to the amount of work required will be charged. When a mounted section is wished for it will be supplied with the charge of one shilling.

It is hoped that those who intend to avail themselves of the Pathological Department of the Journal will read the foregoing remarks carefully, so that they may understand the principles upon which the Department works and the objects of its existence.

In another part of this issue the notice of the Department which will permanently appear in the JOURNAL will be seen. Reports of pathological examinations will not be published in the JOURNAL, but forwarded by post to the sender of the specimen; for this purpose a stamped and addressed envelope must accompany the specimen.

Clinical Lecture on Diabetes.

Delivered at St. Bartholomew's Hospital on Friday,

November 15th, 1895,

By T. LAUDER BRUNTON, M.D., F.R.S.



ENTLEMEN,—It is said that if a man loses himself in a snowstorm, or in the primeval forest, he goes wandering round until he finds himself at the point from which he started. We find very much the same thing in the life of a man, and probably it is this tendency that has led me to choose the subject of "Diabetes" for my first clinical lecture here as full physician of the hospital, as it was the subject that I first took up when I began clinical work in the hospital a great many years ago. The results of my first work were published in the *British Medical Journal* of January and February, 1874, and I have had these lectures reprinted so that each of you may have a copy and consult them, because I have dealt with the subject a good deal more fully there than I could do in the short time at my disposal now.

We find the same kind of tendency not only in individual men, but in numbers of men; and we notice that fashions in dress and in manners come round again and again after the lapse of a number of years. We find the same thing in ideas. The wise man has said, "There is no new thing under the sun. Is there any thing whereof it may be said, See this is new? it hath been of old time, which was before us."^{*}

Now the newest thing in medicine is the administration of tissues and organs in a raw state, either by the stomach or by subcutaneous injection. In place of the raw tissues themselves, extracts of those tissues are frequently employed. This method of treatment is, to a great extent, a new one, and is effecting to a considerable extent a revolution in medicine; yet it is not a new plan. From time immemorial savages have been accustomed to eat parts of the bodies of their slain enemies, especially when those enemies were distinguished by special bravery, or by eating the hearts of the lions or other beasts of prey which they might have overcome, with the idea that by consuming the hearts or other parts of the bodies, either of their enemies or beasts of prey, they would acquire the bravery and power which distinguished their foes.

The plan of treatment by the administration of raw organs was tried by me in this hospital just about twenty-three years ago in cases of diabetes. It seemed to me that some cases of diabetes were due to imperfect utilisation of sugar in the muscles, and that therefore the proper method of treatment would be to supply a substance contained in the muscles which would enable the patient again to utilise the sugar which was present in his blood. This substance I believed to be a ferment which would have the power of splitting up the sugar into the more easily combustible form of lactic acid. I accordingly administered raw meat. I got a certain amount of benefit in some of

the cases, but I obtained no very definite cure. Not finding that I was able to give raw meat in sufficient quantity to gain the effect I desired, I made an attempt to extract from the raw meat a ferment by the method which was then usually employed, viz. solution in glycerine. I noticed, however, that this also was insufficient to effect my purpose, and accordingly the treatment was given up. The method has again been revived of recent years on account of the success that has attended the administration either of thyroid gland or of its glycerine extract in cases of myxœdema. Attention has also been much directed to the subject lately by the researches of Brown-Séquard, who has found that extract of testicles seems to have a tonic effect upon the body generally. In consequence of the results obtained by the extract of testicles and by thyroid gland, researches have been made upon the effect of various other glandular organs. For example, parts of kidney, extract of kidney, extract of suprarenal capsules, extract of brain, and so on, have been given in such cases as seemed appropriate. The results have, however, been rather disappointing, and in none of the other diseases have we obtained any such definite and successful results as in the case of myxœdema.

It is sad in one way, but satisfactory in another, to find that the attempts I made so long ago have even yet been unsurpassed; that the researches that have been made upon the treatment of glycosuria by the administration of various organs have hitherto not been successful, any more than the attempts I made by the administration of raw meat. I find, too, that in my attempt to extract ferment from the muscles I was not successful; but even now physiologists have been no more successful, and in a book on physiological chemistry, which is probably the best and most recent work on the subject, I find the following passage:—"Endlich lässt sich—die Gegenwart eines Milchsäure bildenden Enzymste annehmen. Aber der exakte Beweis für die Existenz dieser Fermente ist vorläufig noch zu liefern" (Neumeister, 'Lehrbuch der physiologischen Chemie,' 2ter Theil, p. 7; Jena, Gustav Fischer, 1895). So that although one is obliged to presume the presence of the ferment, which I believe to be present in muscle, physiologists even now have not been able to show any more than I could the absolute existence of this ferment.

Now, at the time when I wrote those papers upon the pathology and treatment of diabetes, attention was chiefly directed to the nervous system as the origin of the disease, because every one's mind was filled with the striking results of Claude Bernard's experiments on glycosuria consequent upon puncture of the fourth ventricle of the brain. During the time that has elapsed since my lectures were published but very little work has been done in relation to the effects of nervous lesions upon glycosuria, but a good deal has recently been done in relation to the effect of other organs upon the liver and upon glycogenesis. Not only have the experiments of Brown-Séquard, of Horsley, and others upon the testicles and upon the thyroid gland led to the introduction of the system of administering extracts of organs, but they have also drawn attention to a very important function of organs, viz. the internal secretion. The first example of internal secretion which we know of as being regularly recognised by physiologists was the internal secretion of the liver. Until Claude Bernard's time people looked upon the excretion of biliary matters as the chief function of the liver, and when they began to investigate the function of the bile, and found that it had very little or no action in digestion, they began to say that the liver was a fraud, a huge organ, the biggest gland in the body; and all that it could do was simply to excrete a little bile, which was not much good for any purpose whatever. It had, perhaps, a slightly laxative influence upon the bowel, but was not really half so good as a little aloes. So that practically here was the biggest gland in the body, apparently placed in the most prominent position, and yet really useless. But when Claude Bernard took up the function of the liver, and showed that its real use was not for secreting bile, but that it could act as a regulator of tissue change, people began to alter their ideas regarding the value of the organ. He showed that the liver was really what we may term the coal bunker of the body; it gathered up within itself the soluble products of intestinal digestion, it stored them in itself during the periods of fasting, and gave them out gradually to the tissues as they were wanted.

Now this function of the liver is one of the very greatest importance, as you can readily see, because the soluble products of digestion are more or less injurious when poured into the blood in large quantities. It was imagined before that the peptones coming from the breaking up of albuminous tissues were transformed in the liver. We now know that this transformation chiefly occurs in the walls of the intestine; that peptones formed by the splitting up of albuminous tissues during digestion are built up again into some form of albumen in the intestinal walls, but they probably reach the liver in such a condition that they are there also to a great extent

* Eccles. i, 9, 10.

stored up and partially split up. Peptones will also yield a certain amount of glycogen, and an animal that is fed exclusively upon meat will, after a large meal of meat, be found to have stored up a good deal of glycogen in its liver. But the chief function of the liver is to act rather upon the products of the digestion of carbo-hydrates. As you know, starch is transformed in the intestine into sugar, which is soluble and easily absorbed; but this sugar, if poured into the blood in too great a quantity, would be excreted by the kidneys. It is therefore converted in the liver itself into insoluble glycogen, stays there in this form for a certain length of time, and is afterwards gradually changed back again into sugar and poured gently and gradually into the blood, according to the requisites of the various tissues to which the sugar serves as nutriment. Now we find that usually the organs of a man are able to do a good deal more than is required of them. We have two kidneys, although only one would do, as we know from the way in which people get on after excision of a kidney. We have two lungs, but we know that people get along fairly well with one lung, and only a remnant of another. We have only one heart, and we cannot do without it: but the organs which are single, although their presence may be necessary, are rarely worked up to their full extent; and the heart, although its presence is absolutely indispensable to life, is rarely acting to its full extent, and the same thing is the case with the liver. As a rule, the liver is capable of dealing with a great deal more sugar than is sent into it from the intestine; so that if any one of us were to take a very full meal of starch, or even if we were to consume a great deal of cane-sugar, we would probably find very little sugar in the urine, because our livers would be capable of dealing with it. There is, however, a limit to the powers of all organs—to the heart, to the lungs, and of the liver. And if you pour in too much soluble sugar into the stomach and intestines, either of a healthy animal or healthy man, you will be almost sure to find a certain amount of sugar present in the urine, because you have overstepped the limits within which the liver can deal with the sugar. You find the same thing in relation to albumen. Most of us can take a raw egg without any disadvantages, because we are able to digest it, and none of the egg-albumen finds its way through the kidneys. Some years ago Mr. D'Arcy Power and I made a number of experiments on our powers of assimilation. We wanted to get artificial albuminuria in healthy men. I managed to swallow six eggs one after the other. The first was not at all bad,—in fact, rather nice; the second was doubtful, the third I did not care about, and you can hardly fancy how abominable the sixth seemed to be; but Mr. D'Arcy Power managed to swallow twelve. I found that my appetite stopped my taking more than six, and my internal organs were capable of dealing with six eggs, so that I got no albumen in my urine. Mr. D'Arcy Power, having been able to take twelve, overstepped the powers of his intestines and liver, and he got albumen in his urine; you may find that what holds true with egg-albumen holds true also with other forms of albumen; and although I was quite unable to take enough egg-albumen to get it to appear in my urine, I managed to get albumoses appearing in my urine by swallowing a large quantity of beef-tea made from Brand's Extract. My intestine and liver were incapable of dealing with this soluble albumen just as Power's were with white of egg, and so it appeared in the urine. Now the limits of the powers, even of healthy men, vary very considerably in regard to sugar and to albumen, but there are certain cases in which they seem to be lessened. The lessening of the power of dealing with the sugar which is poured into the blood from the intestine tends to produce a form of glycosuria by increasing the amount of sugar in the blood. For if the amount of sugar in the blood exceeds about one-third per cent.—that is to say, .3 per cent.—it passes out into the urine, and gives rise to glycosuria. This imperfect action of the liver in converting the sugar, which is poured into the blood, into insoluble glycogen seems to be the cause of one form of glycosuria.

I would like to point out that just as we formerly included all cases of albuminuria under one general term, "Bright's disease," so we are apt now to include all forms of glycosuria under one general head, that of "diabetes." But you know perfectly well that there are very different forms of albuminuria, some of which are of comparatively little importance, others of which are deadly diseases. And there are very different forms indeed of glycosuria. Some forms are comparatively unimportant, others lead to rapid emaciation and speedy death. You will find that generally glycosuria or diabetes has been divided into two classes according to its causes.

1. Cases of increased formation.
2. Cases of lessened assimilation.

But I think we might divide the glycosuria first of all into two distinct classes. This division has been made already by Harley, who separated cases of glycosuria into the fat and the lean. The fat, how-

ever, may again be subdivided into two distinct classes. I would be inclined to limit the term glycosuria to the fat cases, diabetes to the lean ones. Lancereaux has mentioned that amongst the fat cases you will find glycosuria occurring early in life, about twenty-one in women and twenty-five in men, and the patients suffering from it are often excessively stout. I dare say that in coming up Ludgate Hill you have looked at the picture of Daniel Lambert. Now one does not know—at least, I do not know, but I have very little doubt that Daniel Lambert had glycosuria. You very rarely come across a man so stout as that who has not got sugar in his urine. Now diabetes is generally looked upon as a wasting disease, but Daniel Lambert and men like him have not wasted; and one begins to think what would they have been like if they had not had some sort of safety-valve like glycosuria. If we think of the amount of carbo-hydrates they took, it must have been fabulous. So that in these cases we may call the disease glycosuria, and not diabetes.

These cases of fat people having sugar in their urine at an early age sometimes go on for a considerable length of time. As a rule, they run on for many years, and not unfrequently they may go on past middle age, and occasionally you may find them getting on even to old age. There is another class of the fat kind of diabetes, which I think is perhaps even more markedly to be reckoned as glycosuria, and not diabetes. This class occurs in people about middle age, about forty-five years of age. Sometimes they may be stout; they generally are well nourished and in them we find the symptoms associated with symptoms of gout, and more especially discharge of uric acid. We have not had in the wards a typical specimen of the fat kind at least, but we have had two specimens of what I think was not diabetes, but gouty glycosuria. There was one man especially, who was not staying in the wards, but came up occasionally, a gardener from Swanley. He was a tall man, well built, about fifty years of age, and when I first saw him in the out-patient room he had a large quantity of sugar in his urine. He had begun specially to notice that he was weak, and then became very thirsty; had a very greatly increased appetite, and still became weaker notwithstanding his increased appetite, and the increased amount of food he was able to digest. In him we had no external sign of diabetes at all. He remained well nourished and rather red in colour. In him we found that treatment was very successful, the only treatment that was necessary being to diminish the quantity of carbo-hydrates that he took. We simply cut him off starch and sugar, and in a very short time the urine became perfectly normal, and I hear it remains perfectly normal, no sugar being present in it whatever. There was another man in the wards who was also a specimen of this sort, only he was a good deal younger, and we had hoped to be able to try in his case some of the newer plans of treatment; but our hopes were frustrated, because when the carbo-hydrates were cut off he at once got well, and no more sugar was to be found. There was one point, however, to which I would like to draw your attention in his case, and that is that after the sugar disappeared from the urine there was still a high specific gravity. We were not able to ascertain the exact cause of this high specific gravity, but it reminded me of a case that I examined many years ago, in which after the sugar had gone from the urine inositol was found. It may be that inositol was present in this man, but we do not know because we were not able to get the chemical analysis made, but I would draw your attention to the possible occurrence of inositol in cases where you get a high specific gravity, but where you do not get any increased amount of sugar.

We have, then, in these cases two fairly typical specimens of what we may term "gouty glycosuria" rapidly recovering. In another case we had a different result. A man was admitted with a large quantity of sugar in his urine, with a sore upon his back, and the physical signs of pneumonia in his lungs. Owing to his weak condition a very careful examination of the abdomen was not made. After a short time—in fact, one day after his admission—the sugar disappeared from the urine. In the two former cases the disappearance of the sugar from the urine was a good sign, and they got well forthwith. In this case, where the man was so severely ill, we did not look upon the disappearance of the sugar as a good sign, but, on the contrary, as a sign of probable approaching death, and this turned out to be right, because in two or three days more he died, and we then had an opportunity of making a post-mortem. Both lungs were found to be affected with phthisis, more especially the right; there was a large sore upon his back, sloughing, and having the characters more of a large sloughing carbuncle than an ordinary bed sore. The liver was decidedly cirrhotic. The pancreas was looked at with especial care, because pancreatic changes have recently come to be recognised as very important factors in the production of cases of obstinate diabetes. It seemed a little firm, but there was nothing very definite found to be the matter with it. Sections of it will, however, be made, and we will pay

special attention to the microscopic structure of it, and see whether any alteration can be found. To the naked eye, however, the pancreas appeared to be normal. In this man we had what really seemed to be gouty glycosuria in its beginning running to a very unsuccessful result. Instead of clearing up, as the other two had done, he became worse and died, and yet there did not seem to be anything in the disease itself that was going to carry him off; but wherever you get glycosuria you are always more or less liable to certain complications. The presence of sugar in the tissues seems to lead them to become a more favorable nidus for the presence and growth of various organisms. You know that in cultivating any microbes the addition of a little sugar to the medium frequently makes it a much more favorable ground for the cultivation, so that microbes which would not grow upon peptone broth alone may grow readily by the addition of a little sugar. Now something like this appears to occur in the body, and patients who have got glycosuria are liable first of all to boils and carbuncles, so that the appearance of boils and carbuncles very often leads you to examine the urine and see whether any sugar is present. It would appear that the microbes make their way through the sweat-glands or through the sebaceous follicles into the skin and subcutaneous tissue, and there they grow and multiply, giving rise to boils or carbuncles. You can readily see that what happens in the case of the skin will happen also in other organs,—for example, the lungs. In healthy people if the tubercle bacilli are inhaled, they take such a time to grow that they get expectorated by a healthy man before they are able to get a footing; but if they are inhaled by a man suffering from glycosuria they may find a nidus, grow more quickly, and lead to phthisis, and probably that was what occurred in the patient that we had in the hospital. Therefore in cases of glycosuria you are always afraid of any infective diseases, whatever they may be, and one is afraid of performing operations on persons suffering from glycosuria because, as it is said, the wounds are apt to go wrong. Consequently it is an advantage to your patients and lessens their risks to remove the sugar from the blood and from the urine as quickly as you can, even although the presence of the sugar may not appear to do the patient much harm.

In most of these cases all that is necessary is to prohibit sugar absolutely, and to lessen the starch in the food. A great deal of discussion has arisen as to whether it is advisable to prohibit starch entirely, or to give it in limited quantities. In my opinion it is not advisable to remove carbo-hydrates entirely from the diet; if you do this you find that the patients, as a rule, suffer very much, and that they practically get such an intense craving for carbo-hydrates that they will have them in spite of your prohibition; whereas, if you allow them in a limited form, you may get your patients to stick to the diet as you restrict it, and it is much better that they should take the diet which you have laid down than take the diet which they have laid down for themselves. Generally, you will find that the ordinary diet table for diabetics is this: proteids of all kinds, fish, flesh, fowl, and eggs are allowed in all forms, whether they be fresh or salted or potted, the only prohibition being that the meats or fish should not be taken with sauces containing flour. All green vegetables should be allowed. Frequently such things as Brussels sprouts are prohibited, but if you forbid vegetables of any kind you lessen your patient's dietary very much, and, as a rule, I am inclined to allow fish, flesh, fowl, eggs, all green vegetables,—and, in fact, vegetables of any kind with one or two exceptions. These exceptions practically are carrots, beet, and potatoes. Carrots and beet contain a good deal of sugar, and potatoes contain so much starch that it practically comes to be almost the same as if the patients were taking sugar. Then, again, potatoes may, I think, be sometimes allowed if they are given in one particular way. Diabetics, as a rule, complain very bitterly of their diet, and say that it becomes so distasteful to them. "If you could only give them a little bit of potato they would be so happy." You may allow a little potato if it is given in the form of the potato chips. A single potato taken in that way may be made to fill almost a whole dish. It must be cooked, however, in one particular way, and this is the way. I think it is known to all the cooks on the Continent, but is not known to all the cooks in this country,—indeed, to very few. You must have a large deep pan, not a frying pan, but a pan six inches deep, and this should be nearly full of oil or grease; dripping is as good as any other kind. You put this on the fire, and it comes as you think to the boil, but this boiling is quite delusive. You let it go on boiling, and all the water that is mixed with the dripping boils away, and finally in place of the apparently boiling liquid you get a liquid with a perfectly smooth still surface, which is not boiling at all, and then is the time you are able to cook your potatoes. They should be cut in very thin shavings indeed, and should be then thrown into this practically boiling fat. When I say boiling I mean this very hot fat which does not appear to boil. They are then quickly taken out, and they become under the

influence of the great heat firm and crisp upon the surface, and the fluid that they contain is boiled within these crisp surfaces by the fat, so that they are blown out, and each little shaving of potato, which was originally about as thick as a bit of cardboard, is now about three quarters of an inch thick. These potato chips may be used in cases of diabetes, and you will find that the patients are very fond of them as a rule, and they are enabled to get down a quantity of food that they could not otherwise take.

Of the two classes that we have just been describing, we have seen one class at least and one sub-class, the so-called "fatty" class, and the sub-class "gouty" glycosuria. There is another class which we have not had any opportunity of seeing yet, and that is the thin patients—the true diabetics. These thin patients become rapidly ill; they quickly lose their strength, they become much emaciated, and generally get a very great thirst. Their cases appear to be entirely different from those of gouty glycosuria, because they run a much more rapid course. Gouty glycosuria cases run a very slow course, so much so that I have seen one case, a lady who was over eighty, who had been suffering from gouty glycosuria for over thirty years. She was a well-nourished old lady, and she suffered apparently no discomfort whatever from the disease; and really I was asked to see her not so much because there was anything the matter with her, but because the old lady insisted that she was suffering from a serious disease which lessened her strength, and her strength must be kept up by stimulant, of which she took rather more than her friends thought good for her. She lived on for some years after I saw her, and, I think, died at eighty-eight, although the glycosuria had lasted for thirty or forty years. So that when a patient comes to you with sugar in his urine, you must not at once tell him that he has got diabetes, because diabetes in the minds of your patients means that they have got a mortal disease, and that they are going to die in a few years. True diabetes is a mortal and rapidly fatal disease, killing them off in about three to six years; but the gouty glycosuria cannot be called a mortal disease, because if an old lady dies at eighty-eight you can hardly say it was that which carried her off. Moreover in these cases of gouty glycosuria you will find it is not the glycosuria that is the cause of death except where it leads to complications, as in the case we had in the wards where it led to inflammation of the lungs with tubercle, or where it may lead to any other infective disease, from the readiness with which those germs grow in the tissues containing sugar.

It is well, then, when you get a case of gouty glycosuria to tell your patients that they are having gouty glycosuria and not true diabetes. You should draw a sharp distinction between gouty glycosuria and true diabetes. It is possible that glycosuria may pass into true diabetes, but very often it does not do so, and there is time to warn your patient when the danger really threatens. In the cases of true diabetes we find that we are obliged to be even more strict in regard to diet than we are in the case of those who are suffering from gouty glycosuria. Even then, although the diet is strictly laid down and strictly adhered to, sugar will continue to appear in the urine, and one must do one's best to try and get it utilised.

The remedies that have been used in cases of diabetes are exceedingly numerous, and yet out of the whole of them there appear to be very few which are of any real service. Practically I think we may say that there is only one which is of very great utility, and that is opium and its alkaloids. The alkaloids are codeine and morphine. Codeine is, perhaps, rather more successful than morphine in one respect, viz. that you can push it further without giving your patient so much constipation, or without giving rise to that excessive drowsiness which morphine causes. But morphine has more power than codeine in stopping the sugar. It is not only capable of stopping the sugar when administered in smaller doses, but it will also tend to stop the sugar after you have reached the limit where codeine seems to have lost its effect. It has seemed to me as a rule that patients are able to stand more morphine, or more codeine, when they are suffering from diabetes than when they are suffering from most other diseases, or than ordinary healthy people can stand. In one case I saw codeine pushed up to the dose of 15 grains daily—a large dose,—and this large dose was sufficient to stop the sugar; smaller doses were insufficient.

I have mentioned to you the chief remedy, but there are some other remedies which may be useful, and one of these is salicylate of soda, which I believe to be useful not so much in cases of true diabetes as in cases of gouty glycosuria, and the use of it is rather as a general remedy for gout than as a special one for sugar. I ought to mention here, perhaps, one caution in regard to the diagnosis of diabetes, or rather, I should say, of gouty glycosuria. An old clinical clerk of mine went into practice in the country a number of years ago, and after he had been in practice for about two years he came back to me and said that he was dying. He thought he had got diabetes. He had put himself on a rigid meat diet, and the more rigid he was the

worse he became. I examined the urine, and I mixed it with some Fehling's solution, and sure enough I got down at once a copious precipitate of yellow oxide of copper. I tested it, however, with a bit of litmus paper, and found it to be intensely acid. I then mixed it with its own volume of liquor potassa and heated it, but instead of getting a dark brown coloration as, had there been much sugar in the urine, I would have done, I got no coloration at all. Clearly, then, we had to deal here with a reducing body, but not with grape-sugar. I put a single drop of the urine under the microscope, and it simply crystallised into an almost solid mass of uric acid. The reducing body in the urine was simply an enormous excess of uric acid, and this caused so much reduction of the copper that my pupil believed that he was suffering from diabetes, and, of course, the more meat he ate the more uric acid he got. I told him to put himself on a vegetable diet, and he very soon became well, and has remained so for at least fifteen years. We must, therefore, be cautious in dealing with cases of gouty glycosuria, and not fall into the mistake of confounding them with cases of true diabetes.

There seems to be a loss of power to utilise sugar in all cases of glycosuria, both in those who are stout and those who are thin, and one of the things that we desire most to bring about is the utilisation of sugar in the organisation.

Now although I was unable years ago to cure cases of diabetes either by giving raw meat or by glycerine extract of raw meat, yet it has been abundantly shown that sugar appears to be used up in the muscles; and therefore, if we want to get it utilised, we have to get the sugar circulated freely through the muscles. Dr. Richardson suffered from diabetes himself, and he expresses very strongly the view that exercise is of the utmost utility in glycosuria. He said that at first when you begin to take exercise you will find that it is very tiring, and that you wish to lie quiet; but if you go on in spite of your languor you will find that the languor will pass off, and after a while you will be able to walk with considerable freedom, and to take pleasure in the exercise, and at the same time feel very much better for it. But in all cases there is a difficulty about taking active exercise. In the case of remarkably fat people, they cannot take exercise because their bodies are too heavy. You will sometimes see this at Marienbad or at Carlsbad, where a lot of those very fat patients travel in the luggage van because they are absolutely unable to get into any of the carriages. I saw one at Marienbad towards the end of the season who was able to walk about, but when he arrived at Marienbad he was in a very bad case, because there was not a single vehicle at the station that would take him for fear of breaking the springs, and he had to get some sort of heavy cart to convey him to his hotel, and then he had a special one built, in which he was carried about. In cases like this it is absolutely impossible for our patients to walk. In the case of the very thin, nervous patients, they are unable to walk because the exhaustion is so great. So that in all cases what we wish to do is to bring the blood freely into contact with the muscles, to allow it to circulate freely through them, and this we are able to do by the plan which has been advocated so largely by Weir Mitchell, viz. by massage. It was found by Ludwig and some of his scholars that if you stimulate a muscle you increase the blood-flow through that muscle. It seemed to me likely that the same result would be got from massage, so Dr. Tunnicliffe and I made some experiments, and we found that after the massage of a muscle the blood rushes through it three times as quickly as before. It may not seem very much to you to hear that the blood travels through the muscle three times as quickly as before, but when you see the blood travelling along two inches only in a glass tube in one minute before massage, and after massage six inches in the same time, it then strikes you as a very remarkable increase. In consequence of this effect on the circulation, in cases where patients are unfit, either because they are too fat, or because they are too weak to take exercise, you may substitute massage in bed for the exercise. There are many other points I should like to bring before you about this disease, but the time is nearly up, and I would only reiterate those things that I want you especially to notice.

I want you first of all to notice that there are really two distinct diseases in which you find sugar in the urine. There is glycosuria, generally gouty; and there is true diabetes. Glycosuria is associated with a well-nourished body, sometimes with a tendency to great deposit of fat; it runs a slow course; it may not kill the patient at all, although it does render him more liable to death from infective diseases. Then there is true diabetes, in which you have the patient thin and weak, generally with greatly increased appetite and with very great thirst. Both of these symptoms may be completely absent in cases of gouty glycosuria, although you generally find them thirsty, but still there may be no greatly increased thirst and no greatly increased appetite. In the case of true diabetes you find the patients

are subject to great wasting. The course of the disease in true diabetes is a short one. It does not, as a rule, run for more than three to six years; although gouty glycosuria may run on for thirty or forty years, as happened in the case of the lady I referred to.

The next point is that I think it is not advisable to be too strict in the regulation of diet. You ought to be strict in cutting off all sugar, and in reducing the carbo-hydrates, such as starch, almost to a minimum; but if you cut them off entirely you run the risk of getting your patients taking more than you would allow, whereas if you let them have a certain amount you may manage to keep them to the restricted diet.

The next thing is that the best remedy in gouty glycosuria is exercise, and if the patients cannot take exercise they can have massage, and you may give them salicylate of soda; but in cases of true diabetes almost the only remedy we can trust to at all is the administration either of opium or one of its alkaloids.

The Causes and Treatment of Nasal Obstruction in Children.*

By ANTHONY A. BOWLEY, F.R.C.S.,

Assistant Surgeon to the Hospital.

(Continued from page 56.)

ADENOID GROWTHS AND ENLARGED TONSILS.



Now come to those cases where there is nasal obstruction due to adenoid growths in the naso-pharynx, and in many of these cases there is also nasal discharge due to some hypertrophic rhinitis and chronic nasal catarrh. The symptoms of adenoid growths are now so well recognised that I need do little more than enumerate them. The child, in a typical case, breathes heavily, and usually keeps the mouth open; there is inability to breathe through the nostrils, and constant snuffling; the voice is deadened and thick; the facial expression is stupid; the child snores loudly, sleeps restlessly, and wakes frequently, and occasionally a little blood is found on the pillow-case in the morning; some degree of deafness is present, and is much worse if the child catches a cold, whilst in many cases of long standing there is discharge from the ear and perforation of the tympanic membrane; the child is undersized, and the general health is not good. It is seen on examination that the nostrils are small and collapsed, and do not dilate on respiration.

But, whilst all these conditions may be present in a severe and typical case, it is not to be supposed that they are always found in every case, and it is certainly true that many patients with adenoid growth are quite as well grown and robust as other children of their own age, and that, whilst other symptoms are present, deafness may be absent. Too much stress must, therefore, not be laid on the concurrence of all these symptoms enumerated.

It seems to me, however, that whilst there is now not much danger of adenoid growths being overlooked, there is a tendency to diagnose them when they are not present, and to advise operations when none are necessary. You may, perhaps, think that this is a strange assertion to make, but I wish particularly to point out that in a large number of cases of nasal obstruction, with many of the symptoms of adenoids, the obstruction is due to hypertrophic rhinitis, to chronic nasal catarrh, or even to purulent rhinitis. I have frequently seen such cases, and have on various occasions declined to operate upon patients who have been sent to me for operation with the diagnosis of adenoid growths, and it is because of the liability to overlook these conditions, and to consider every case of nasal obstruction as one of adenoids that I ventured to first of all place before you a few details of the varieties of nasal catarrh.

I would next point out that errors also arise from an examination of the pharynx with the finger, and the discovery of some swelling in the posterior wall of the naso-pharynx. Adenoid growths are, after all, merely an increase of the normal lymphatic glandular tissue of this region, and a certain amount of swelling is quite a natural thing in this situation. It is only when this is excessive and is associated with swelling and congestion of the neighbouring mucous membrane that an abnormal condition can be said to exist. It follows, therefore, that no question of operation is to be considered merely because

* A paper read before a meeting of the North-Eastern Division of the Metropolitan Counties Branch of the British Medical Association.

there is some perceptible swelling in the naso-pharynx, and that unless there are associated symptoms certainly no operation is to be performed. I mention this particularly because there seems to me at present to be a decided tendency to advise an "operation for adenoids" in many cases where none is needed, and the error seems to be due to either overlooking some other cause of nasal obstruction, or to putting too serious an interpretation on the discovery in the pharynx of a collection of adenoid tissue which is a natural constituent of the mucous membrane in this region.

The next matter deserving of attention in deciding whether or no an operation is to be advised is that the size of the adenoid growth bears no direct relation to the severity of the symptoms. Quite a considerable growth may cause but few symptoms if no catarrh of the mucous membrane accompanies it, whilst quite small growths may cause severe symptoms if they set up and maintain catarrh. I should say, therefore, that if there are definite symptoms of nasal obstruction in association with even small adenoid growths, especially if these symptoms are associated with increasing deafness, it is certainly advisable to operate, supposing no other cause of the trouble be discovered on examination. I should, myself, lay especial stress on the presence of deafness, and in any case of doubt would operate for this alone if it could not be improved by other treatment. I have come to this conclusion gradually, because I have noticed that in several cases where I had removed but very little growth the deafness and other symptoms have rapidly disappeared, and in some of the cases where I have operated solely because of the deafness and in the absence of almost all other symptoms I have found definite abnormal growth, and the deafness has at once improved. I am, indeed, quite sure that in many cases deafness may be the only material symptom resulting from adenoid growth, but by itself it is quite sufficient to warrant the removal by operation of any growth that is present, so as to save the patient from a lifelong affliction.

With regard to the operation itself, it seems to me that there is a tendency on the one hand to greatly exaggerate its difficulties and dangers, and on the other to assert that the operation is so trivial as to be of no importance. I think the truth is midway between these extremes. No operation can be said to be absolutely trivial when fatalities have occurred in the practice of the best surgeons, and to approach any operation in such a spirit is to court disaster. On the other hand, the operation is not at all a serious or dangerous one if all proper precautions are taken, and it does not present any serious mechanical difficulties, and is quite as easy with a little practice as a great many other minor operations.

Perhaps the next question to consider is that of an anæsthetic, and I have no hesitation at all in expressing the opinion that chloroform, or a mixture of chloroform and ether, is the best. I have tried both gas and ether, and do not like either as well as chloroform; my reasons being that both of them cause great venous turgescence and increase in the hæmorrhage, and that the patient is liable to recover from the anæsthesia too soon if the case be a troublesome one or complicated by enlarged tonsils. It is, however, quite unnecessary to produce deep anæsthesia with chloroform, and further, it is certainly unsafe to do so. The operation should be commenced as soon as the child is quiet enough to admit of the finger being passed into the pharynx without struggling, and this is long before deep anæsthesia is induced. If there is much tendency to vomit, it is best to encourage it by tickling the throat, so as to get rid of any mucus before commencing the operation. Particular care should, however, always be taken that the stomach is empty, and that no solid food has been taken for three or four hours prior to the removal of the growths, though in all children in whom the operation is not done early in the morning a cup of strong soup or beef tea may be given with advantage an hour or two previously.

As to the mode of operating, I would first of all say that mere scraping with the finger-nail is quite inefficient except in a few trifling cases, and is never sufficient for the satisfactory removal of growths of any size. For the same reason I object also to the artificial steel nail introduced by Sir William Dalby, which is further impossible to use in the pharynx of a small child, when sometimes even an unnumbered finger may find difficulty in passing. The other methods which may be adopted are the use of some form of curette or of forceps. The growths may be satisfactorily removed with either, but I do not myself like curettes, for when the growth has been scraped away by them it is left in the throat, and may possibly enter the larynx, especially if it be left partially attached and hanging by a strip of mucous membrane.

Forceps, on the other hand, remove the growth clear of the mouth, and are in all ways suitable for the extirpation of the growths. I have for some time used a slightly modified Lowenberg's forceps, made so that the blades cross one another like scissors, and do not

meet edge to edge, the result being a much improved cutting instrument.

The position I prefer is a recumbent one with the head a little thrown back—but not hanging back—and turned to one side, as this position enables the operator to see clearly the back of the throat, whilst at the same time it facilitates the introduction of the forceps, allows the blood to flow into the cheek, and prevents its collection in the pharynx. In seizing the growth the back of the forceps should be kept closely applied to the pharyngeal wall, and at the same time care should be taken not to tilt the blades so as to cause them to point towards the posterior nares. Three or four introductions of the forceps is usually quite sufficient, and as two minutes is an ample time for the removal of the growths, no chloroform need be given during the operation itself.

In my opinion there is never any growth except in the posterior wall of the pharynx that requires removal by forceps, but after the latter have done their work the naso-pharynx and the posterior nares should be thoroughly scraped with the finger-nail, so as to remove any thickening that has escaped the cutting blades. Many operators speak of the necessity of scraping growths from the neighbourhood of the Eustachian tube, and various instruments have been invented for this purpose. I am quite convinced myself that such operations are not only needless, but are harmful, for the thickening around the tube, which only sometimes exists, is due merely to congestion and œdema, and not to adenoid growth, and will all subside when the latter has been removed. Scraping the mucous membrane around the orifice of the tube with sharp instruments is in my opinion both an unnecessary and dangerous treatment.

The amount of bleeding that complicates this operation varies very greatly, but it never continues for more than a very few minutes, and then rapidly and spontaneously ceases. No treatment is ever required to arrest it in my experience. It is, however, of great importance to mop up the blood that flows into the pharynx, and occasionally, though rarely, it is necessary to stop the operation for a few moments to clear it away. When the operation is completed care should be taken to keep the patient on the side, so that if any blood which has been swallowed be vomited, it may be at once got rid of. The subsequent treatment consists practically in leaving well alone, and keeping the child in bed for two or three days and in the house for a week, so as to avoid all chance of catching cold. On no account should the nose be syringed, as this is more likely than anything else to cause an attack of aural catarrh with possibly suppurative. It is sometimes important to mention this to parents, for many of these children have previously been treated by syringing, and this is likely to be resorted to if there are complaints of the nose being blocked up.

I fear I may have wearied you with the details I have described, but my excuse must be that success depends upon details, and can only be ensured by constantly keeping this in mind. But tolerably uniform success can so be attained, even in out-patients' hospital practice, is sufficiently demonstrated by the fact that during the past three years more than 600 patients have been operated upon by myself or under my supervision in the Department for Diseases of the Throat at St. Bartholomew's Hospital, without serious complication of any kind occurring during operation or convalescence. And when it is remembered that many of these children are placed in very insanitary surroundings at home, I think we may well be surprised at such a result. It is, further, evident that if patients can do so well under these circumstances, we may feel sure of obtaining equally good results at least in private practice.

With regard to the effect of operations upon the symptoms that have been produced by the adenoid growths, I think it may be said that when the patients are children the prognosis is very good. Parents must, however, be warned that the deafness, the nasal intonation of voice, and the habitual open-mouthed expression will not all disappear at once, and that in older children the last two generally require careful training for their eradication. If deafness continue for more than a few weeks it should be treated by the occasional use of a Politzer's bag.

May I now turn for a very short time to the subject of *chronic enlargement of the tonsils*? for it so much associated with the growth of adenoid tissue in the naso-pharynx that I find it difficult not to mention it in this association.

With regard to the enlargement itself, it is often, in my opinion, a true hypertrophy, a genuine increase and enlargement of tonsillar tissue; in other cases the enlargement is inflammatory, and results in part at least from frequent attacks of tonsillitis of very various intensity. Now I feel sure that there are very vague ideas as to the natural size of the tonsils in young children, and should like to point out here what I find it advisable to demonstrate at the Hospital,

that in little children under the age of three or four quite normal tonsils can be made to project so as to appear very greatly enlarged, — and even almost to meet — if the child strains, as it so often does, as soon as a spatula is put into its mouth.

If an inspection be made whilst the child strains or retches, a diagnosis of enlarged tonsils may easily be made when no enlargement exists. This is all the more likely to be the case because the tonsils are normally much larger in proportion to other parts of the mouth in children of tender years, and what would be relatively a large tonsil in a boy of ten is a normal structure in a child of three.

I mention this because I find that some students seem to me to find enlarged tonsils in most of the children they examine, and I think what I have mentioned may possibly be a source of error. But, keeping such facts in mind, it is generally easy enough to say whether tonsils are unusually large or not; and when they are very greatly enlarged, so as to block the pharynx, there can, I think, be no doubt that they should be removed.

I do not myself advise operation unless, first, symptoms of obstruction to respiration exist; or, secondly, the patient is the subject of recurring attacks of tonsillitis. Without one or other of these troubles, operation is not necessary.

Let me next say that in the first of these classes, namely, where there is obstruction to respiration, I feel sure that there is practically always in young children some adenoid growth as well, and, as that growth is often present in the second class also, I always arrange to operate on the tonsils under an anæsthetic, and to remove any adenoids at the same time. I should, nevertheless, like to make it clear that in older patients enlarged tonsils requiring removal are often unassociated with naso-pharyngeal growth, and that although when enlarged tonsils in young children require removal there are also, as a rule, adenoids to remove as well, it is not true that in all or nearly all cases of adenoids the tonsils are enlarged. I think also that in operations under an anæsthetic the tonsils are really more satisfactorily removed than when a young or frightened child is screaming and struggling. The operation of tonsillotomy in young children is thus, as a rule, to be associated with removal of adenoid growths, and in general terms the same precautions as those already described should be taken. My own practice is to remove the tonsils first, and to allow the escape of any freely flowing blood for a minute or so before going on with the adenoids; but if there be no material hæmorrhage there is no need for any such delay. As to the mode of removal, I suppose that each surgeon will do best the operation he is most accustomed to perform, but I may say that I prefer the tonsillotomy that goes by Mackenzie's name.

The subject of hæmorrhage after tonsillotomy is not of quite so simple a nature as that of the bleeding which occurs after the removal of adenoids, and it is a matter on which both surgeons and the public feel some little anxiety. I always console myself with the consideration that up to the present time there are no recorded cases of death from hæmorrhage after tonsillotomy in a child, so that when I see a good deal of bleeding I consider that after all this is not likely to be the first fatal case. I think, however, it may be said that the older the patient, the more likely is there to be hæmorrhage, and in adults it is quite certain that fatalities have occurred, so that the nearer the child is to puberty, the more likely is this bleeding to be smart.

May I next say that I consider some of the routine treatment of these cases quite erroneous, and that I have not yet personally had to do with any case in which bleeding has not spontaneously ceased in children? The erroneous treatment I allude to is putting the patient in the sitting or even standing position, and encouraging him to gargle with cold water, to suck ice, and to spit out the blood. If you watch a patient acting thus you will see that the bleeding parts are never kept quiet for a moment, and in his efforts to get rid of the blood from the pharynx the raw surfaces are squeezed by the contraction of the pharyngeal muscles, and are made to bleed the more. I believe that if there is considerable hæmorrhage it can be made to continue for an almost unlimited time if the patient is only supplied with a basin to spit into and something to gargle.

You will gather from what I have said that I prefer the patient kept quiet, and whether an anæsthetic has been administered or not, I should always insist on the recumbent position and perfect quiet as the first essential for the arrest of the bleeding. I can further say that I have never in children required to adopt any other treatment when rest and quiet have been insisted upon, although some oozing may continue for half an hour or more. In hospital practice I never send a child away until the bleeding has definitely ceased; if it continue I take it in for the night and put it to bed. In private practice I always prefer to operate on children in their own homes, because it is not convenient to put them to bed in my own house; but I would especially insist on the fact that bleeding will almost invariably cease

spontaneously if the patient be kept at rest in the recumbent position, and is not encouraged to gargle or to spit. If you send a patient walking home for some distance after tonsillotomy he may lose a great deal of blood *en route*, and I have had to treat on several occasions children whose tonsils have been removed outside St. Bartholomew's, and who have come there blanched from loss of blood. Here, again, the first thing is to put the patient to bed. To further prevent secondary hæmorrhage I think it wise to insist that no hard food of any kind should be taken for a full week.

But although I am of opinion that in children bleeding may almost always be arrested by such simple treatment, I am of course aware of recorded cases where there has been difficulty in stopping the hæmorrhage, and I have myself had to do with older patients where other means have had to be adopted. The first and most efficient of these is, in my opinion, direct pressure; and often during an operation, when the bleeding seems likely to cause delay, I compress the bleeding surface with a small sponge or piece of wool applied to the bleeding surface with the tip of the index finger, and generally with some immediate improvement. The same treatment may be continued, with a gag to protect the operator, if bleeding continue in spite of rest, or the pressure may be applied with a sponge on a pair of forceps. I am glad to say that beyond this my personal experience does not extend, for I have not yet had to employ any other means. If, however, I were pressed as to what I should do in case this proved inefficient, I think I should either apply some dry sulphate of iron to the bleeding part with a small piece of sponge, or should touch the bleeding point with a cautery at a dull red or black heat. I should suppose also that in such cases the use internally of such drugs as ergot, gallic acid, opium, or hamamelis might prove beneficial, and I should certainly try them. As to tying any vessels in the neck, it is scarcely necessary to discuss that matter at present, for ligation of the external carotid has not yet been required in any case in a child, and I do not think it ever will be if ordinary precautions are taken.

And here I will leave the tonsils, for I think I have nothing to add with regard to tonsillitis in its more acute forms than is already well known. But before I bring this paper to a conclusion altogether I should like to say a very few words on the subject of tumours of the nose and naso-pharynx in childhood. They are fortunately very rare, for even the common nasal polyp does not usually commence till childhood is nearly passed and the period of puberty reached, although in cases of watery discharge and apparently simple chronic catarrh it must always be remembered that these symptoms may be due to the presence of polyps. Again, when the naso-pharynx is blocked, it must not be forgotten that the fibro-sarcomatous and fibro-angiomatic growths of this region usually develop in children, and generally in boys of from ten years and upwards. They may in their earlier stages closely simulate the symptoms of adenoid growths, but they are fortunately much more rare, and usually require considerable operations for their removal. There are practically no other tumours of the nose or naso-pharynx that require mention, and I have merely alluded to these two classes because it is well to remember that they may simulate other and less serious conditions.

Case of Acute Tubercular (?) Peritonitis with Alarming Symptoms; Recovery after Paracentesis only.

By SAMUEL WEST, M.D.



ON August 15th, William R—, æt. 15, was admitted into the hospital in an almost moribund state, with a temperature of 104°, and great distension of the abdomen. The history that the patient gave was as follows. It appeared that he had been in his usual health until the 12th of August, when he had a little diarrhœa. On the 14th his abdomen suddenly swelled, and he had a great deal of pain in it. On the 15th (the day of admission) the abdominal distension was considerable, and the bowels had not been open for twenty-four hours, though there had been no vomiting.

The patient appeared to have been a fairly strong lad, and there was nothing at all worthy of note in his family history. One month previously he had had a fall on the back of his head, which had stunned him, and he had been unable to see clearly for some hours, but there seemed to be no ground for connecting this fall in any way with his present illness.

The lad was well nourished and developed, and looked as if he had been in fairly good health previously. He now looked extremely ill, face very pale, his pulse 110, of low tension, his temperature between 103° and 104° , and his skin slightly sallow but not dry. His general appearance was rather that of collapse than of acute pain. He preferred to lie upon his back, and his legs were often not drawn up. The tongue was dry, the abdomen very large, tense, and measured twenty-eight inches in circumference. The subcutaneous veins were enlarged all over its surface, especially in the flanks, but the blood ran equally in either direction. The abdomen was only slightly tender on palpation, but there was no marked pain. It was resonant all over except in the right flank, where the percussion was somewhat impaired, and this impairment disappeared when the patient was turned on to the opposite side. An indistinct thrill was also thought to be felt, so that it was probable there was fluid present in the peritoneal cavity. The respiratory movements, although diminished, were not completely absent. Examination of the rectum showed nothing, but some scybala were found. The distension of the abdomen had displaced the organs somewhat upwards into the thorax, but otherwise there were no symptoms or physical signs in the chest. The patient was desperately ill and evidently in a very critical condition, and it was thought desirable to send for me in the middle of the night.

I thought it evident that the case was one of acute peritonitis, but I could not satisfy myself as to its cause. The question I had to decide was whether the abdomen should be opened or not, and after careful consideration I decided against operation, because the patient was so desperately ill that I hardly thought an operation could be successful in his then condition, and because it seemed to me that the case could safely be left for a few hours, that is to the next day, when the question might be reconsidered, and when possibly the patient might be in a somewhat better state, for worse he could hardly be. On the whole the case presented the appearances most of perforative peritonitis, but with what organ it was originally to be connected it was impossible to say, for there was nothing in the previous history or in the physical condition to assist us in the diagnosis.

The patient was ordered to be fed with small quantities of milk and brandy at a time. As the patient was suffering no particular pain no opium was given or other drugs.

The next day the temperature had fallen in the morning to $99^{\circ}8'$; and the patient was certainly no worse, but rather better. He had taken three ounces of essence and an ounce and a half of brandy during the night; his pulse was rather stronger, and his general strength maintained.

In the afternoon physical examination showed that there had evidently been a considerable increase in the amount of fluid, although the girth of the abdomen was not very much increased. This rapid increase in fluid was thought to be against the notion of its being purulent.

The patient passed a natural motion in the evening, and two more motions the next morning. On the following day, August 17th, the fluid had still further increased, and paracentesis was now performed, with the result of removing four pints of serous fluid. The intra-abdominal pressure was high, viz. eight inches of water, and there was a respiratory oscillation of half an inch. The temperature oscillated during the twenty-four hours between 99° and 101° . The operation gave great relief.

The further history of the case is one of gradual recovery. The fluid gradually disappeared from the abdomen. The temperature continued hectic for three weeks, oscillating daily between normal and 102° , after which time it became normal. The abdomen presented no marked abnormality, except that it was a little pulpy and boggy to the touch on leaving the hospital. The case would probably have been diagnosed as one of chronic tubercular peritonitis, but only if attention had been specially directed to the abdomen. No history of any previous illness was obtainable, and the abdomen had not been observed to be getting larger before the present attack.

In the end there could be no doubt as to the diagnosis, viz. tubercular peritonitis; but the case was one of great interest at the time, and I think unusual. Effusion as a rule develops slowly and gradually in chronic tubercular peritonitis; and although it is not uncommon to see an effusion form in a few days so as to require paracentesis, still it is, I believe, quite rare to see an acute onset as in this case with all the signs of acute general peritonitis, attended with such urgent symptoms in two days' time as to place the patient in immediate danger to life.

It is difficult in verbal description to say why I thought operation undesirable when summoned to the case on the night of his admission. Every one agreed that the patient's life was in imminent danger. Some argued—the patient is bound to die if left alone; operation may relieve, therefore an operation should be performed. I felt that the

patient was so ill that operation would probably kill him, and that it would take away any chance he had of fighting through. The child was greatly collapsed from the result of his removal to the hospital, and I thought that, desirable as it is if an operation is to be done at all to do it without unnecessary delay, still in this case a few hours longer could hardly make him worse, and might make him better. The operation then, if it proved necessary, would be performed under more favorable conditions. The result fully justified the view I took, for the patient got well without operation. Yet every one agreed at the time that opening the abdomen in his then condition would in all probability be fatal.

Physicians naturally look on these cases from a different point of view, and with somewhat different experience to guide them than surgeons; and though approving heartily of the general rule of operating early in all cases which require it, and accepting the general statement that as a rule more harm is done by delay than by operation, still these are only general rules, and there are exceptions to each of them. Yet in the right kinds of cases as much wisdom may be shown in refusing to operate as in advising operation, perhaps more, and this was emphatically a case in point.

The Mechanism of Phagocytosis.

A Paper read before the Abernethian Society on

November 14th, 1895.

BY W. LANGDON BROWN, M.A.,

Assistant Demonstrator of Biology.

(Continued from page 42.)

III. The degrees of excitability of the Sporadic Mesoblast; the functions served thereby.

In Richet's* definition of irritability we find the following generalisation:—"The movement in response to irritation is, for equal irritations, stronger in proportion as the equilibrium of the cell is less stable; in other words, stronger as the cell is more excitable." The wide differences of stability found among the cells of the sporadic mesoblast afford many illustrations of the truth of this deduction.

(1) The most unstable wandering cell of all is that originally described by Hardy† in the cray-fish, and appropriately termed by him the "explosive cell." To see such cells the blood must be examined within a few seconds from the time of its removal from the animal. A large number of pale oval corpuscles are seen to shoot out extremely fine pseudopodia, along which blebs of cell substance travel rapidly, expand into a vesicle and burst. Or vesicles may develop directly from the surface of the cell, and expand until they burst. Normally these cells are actively motile and ingestive. If a quantity of Indian ink or milk be injected into the animal they will rapidly take up the particles; and their avidity for ingestion is on a level with their explosiveness, for they will include a particle so large that the cell can form but a fine film around it. After ingestion they will be found to have lost much of their explosive power. Apparently their stability is increased. Applying the results obtained in the previous section, we may see that the cell explodes chiefly from the stimulus of contact with foreign bodies, to which it attempts to offer the greatest amount of surface; its irritability is such that the expansion takes place almost instantaneously. But if the cell has already "imploded," as it were, around a particle of milk, it cannot react nearly so violently to the stimulus of another foreign surface, as it is already reacting to the included particle; hence explosions occur more rarely and slowly. The best method of preserving these cells is by the immediate application of a drop of iodine solution. It is of interest to note that the "fibrin-ferment" appears to be stored in these explosive cells, for crayfish's blood ordinarily becomes solid immediately after shedding; it clots just as rapidly as the explosions occur. Now iodine solution '25 per cent. will delay the explosions, and clotting is deferred by just the same length of time.

It is probable that explosive cells might be found in many forms of life if only search were made. When working on the blood of Molluscs I found that the Lamellibranch Unio would yield admirable instances of explosive cells; in fact, explosions occurred so rapidly that the only way to make satisfactory preparations was to draw the blood direct into a pipette containing the iodine solution, so that as

* *Essai de Psychologie générale.*

† *Journ. Phys.*, vol. xiii.

soon as the cells came into contact with any foreign matter they encountered the preservative agent.

An explosive cell with coarse basophile granules has also been recently found by Kanthack and Hardy (loc. cit.) in the guinea-pig and rabbit. The specially rapid modes of preparation necessary to see such cells has naturally led to their being overlooked. Thus Geddes* describes in the perivisceral fluid of sea-urchins forms which strongly suggest that he was looking at explosive cells which had burst before he began his observation.

(ii) The next most irritable wandering cell we meet with is the one assisting in the formation of a plasmodium. A plasmodium, we may say at once, seems in invertebrate blood to take the place occupied by the clot in vertebrate blood. When the blood for instance in Molluscs and Echinoderms is shed the ordinary non-granular cells, throwing out pseudopodia and thin sarcoidal plates, soon meet the corresponding processes of neighbouring cells. By this time these processes have begun to form a continuous zone or stellate crown around each cell, and as they unite together the individual cell-outlines become progressively less distinct. Thus a plasmodium is formed into which the granular cells never enter except by passive inclusion.

(iii) Thirdly, we come to the ordinary phagocyte, which stands next in order of irritability; it is more sluggish than the two types just described, but, as we know, is normally amœboid. In fact its method of inclusion is precisely that of the amœba, which has been carefully described by Miss Greenwood,† as taking place in two stages: (a) two pseudopodia spreading round the foreign body meet on its further side and join together; (b) the foreign body is then closed in above and below by the ectosarc being drawn over it like the rim of a funnel. Thus the prey lies completely enclosed in a vacuole. Whatever be the exact nature of the stimulus, it is of interest to see that the reaction will continue after the apparent cessation of that stimulus. Thus an active infusorian may escape while the encircling pseudopodia are but half advanced. Yet the advance of the pseudopodia is continued, despite the fact that the prey has fled, and ultimately they meet round a space containing only fluid.

(iv) All the foregoing types are normally mobile. But there are some cells which, belonging to the sporadic mesoderm, need abnormal stimuli to call their amœboid nature into play. Such a one is the clasmotocyte of Ranvier already described, which having settled down and inserted its long ramifying processes into the crevices of areolar tissue will not stir until stimulated by inflammation, when it rapidly buds off wandering cells. Another example is the large phagocyte described by Heidenhain in the intestine of the guinea-pig, which, normally stationary, will, if the animal be starved, pass from the parenchyma of the villus into the epithelium, which it proceeds to devour.

We have seen that no hard-and-fast line can be drawn between such sluggish forms as these on the one hand and the explosive cell on the other. By increasing the stimulus one type will imitate the ordinary properties of another more irritable type. Thus the blood-corpuscles of *Daphnia*, not ordinarily explosive, become so at once if brought into contact with the poisons produced by the *Torula* occasionally parasitic in its blood (Metschnikoff). And the white blood-cells of a frog require, as we have said, gentle heat before they will throw out the long pseudopodia and delicate plates normally seen on shedding invertebrate blood.

(v) Just in the same way we find cells which are normally definitely fixed becoming contractile and even amœboid on being stimulated to an unaccustomed degree. Thus Metschnikoff‡ has shown that in the caudal fin of Triton or the tail of a tadpole the fixed connective-tissue cells in the neighbourhood of an inflammation become vacuolated, while their processes become shorter and lose many of their branches. Ranvier,§ too, has recently described how during inflammation of the great omentum: long processes develop from the cells of the connective tissue; these grow along the threads of the fibrous cobweb formed by a blood-clot; they meet corresponding processes from neighbouring cells, and fuse with them. In this way protoplasmic bands are rapidly formed. Ranvier believes this plays an important part in union "by first intention," where the first steps take place too soon to be accounted for by proliferation of connective tissue, which does not occur till towards the close of the third day following the injury.

Some of the most striking instances of fixed mesoblastic cells becoming mobile again are to be found in the behaviour of the

capillary endothelium in inflammation. Embryologically formed from mobile cells, under abnormal stimuli, it may show this ancestral trait again. Severini* even claims that this characteristic normally plays a part in regulating the calibre of capillaries. According to him oxygen causes them to adopt a more spherical condition, so that they bulge into the lumen of the capillary and constrict it, while carbon dioxide causes them to flatten, thus leading to dilatation of the capillary. This seems somewhat doubtful, but it appears probable that the stigmata through which the leucocytes escape in inflammation originate from contraction of the endothelial cells. Even these have been referred, however, to the passive expansion of the vessel, resulting in a series of leaks, as it were, between the cells.

A good example of the vicarious phagocytic action of the endothelium is seen on intravascular injection of bacilli. This may be followed by proliferation of the endothelial cells, leaving the adventitia bare; such proliferated cells even enclosing pigment and granules. Another example is seen after death from malaria, when the endothelium of the hepatic vessels is found filled with the characteristic parasite. Pathology yields suggestions of a similar process affecting epithelial cells in many catarrhal inflammations.

The question naturally arises next—Do amœboid cells ever become elements of the fixed connective tissue? Round this point controversy has raged fiercely, and we must deal with it very briefly. Cohnheim and Ziegler both attributed an important part in the repair of tissues to the migrated white blood-corpuscle. In 1875 this was the generally accepted view, but each subsequent observer "has more pronouncedly tended to deny to leucocytes an exclusive share in the replacing of coagulum, &c., by cicatricial tissue." The method adopted for the study of this question has been to introduce into the peritoneal cavity or subcutaneous tissue of an animal a little glass chamber formed of two circular cover-slips between which a strip of tinfoil is cemented round eleven-twelfths of the circumference. Thus the tiny cell has an opening into which wandering corpuscles enter between the two ends of the tinfoil strip. At varying intervals the cells can be removed and examined microscopically. Sherrington and Ballance,† in carefully repeating these experiments, could not satisfy themselves that the leucocytes which immigrated into the cell ever gave rise to connective tissue. The leucocyte appeared to fall a prey to the plasma cell of the connective tissue, which in its turn gave rise to the "fibroblasts," forming the new connective tissue. They pointed out that "it is quite in accord with the laws of natural descent for the cells of connective-tissue, when thrown into renewed and extraordinary genetic activity . . . to produce a progeny of cells possessed of the same tendencies as themselves." So that in warm-blooded animals at least the leucocyte does not appear to settle down as an element of fixed connective tissue. Putting it in general terms, a fixed mesoblastic cell becomes sporadic for a time under an abnormal stimulus, but soon reverts to its fixed life again. So far has such a view become current that at the International Congress of Physiologists held in Berlin in 1890 Ziegler publicly recanted from his former position. Metschnikoff (op. cit.) still holds the contrary view; and it may be that in cold-blooded animals the leucocyte takes part in the processes of repair. He states that in the tadpole's tail in which an injury is being repaired the so-called polynuclear cell undergoes a fusion of its nuclei, or (as we should say now more accurately) by a condensation of its nucleus, and then settles down into a typical branched connective-tissue cell. If the wound be inflicted with an instrument powdered with carmine the leucocytes ingest the coloured particles, which are afterwards found in the interior of the connective-tissue cells. And he maintains that a cell once fixed never ingests foreign matter. A negative is notoriously difficult to prove; remnants of intracellular digestion are found among fixed cells, and this may be an instance. Where such authorities differ who shall decide? We can only say that in warm-blooded animals the part played by leucocytes in the formation of new tissue is not proven, and in cold-blooded vertebrates such is not the usual agency employed in repair.

We have traced then a series of cells, some always wandering, some, though naturally amœboid, adopting a fixed position for the major part of the time, and others usually fixed but becoming amœboid with specially strong stimuli. It is a scale showing increasing stability in the cell; and we have seen that "the response to irritation is . . . stronger in proportion as the equilibrium of the cell is less stable."

The irritability of the cell having manifested itself in one way, it naturally tends to react much more sluggishly to any further stimulus. Thus we saw that in the crayfish if the explosive cells have previously ingested milk or particles of Indian ink, they are much less prone

* *Arch. de Zool. Exp.*, viii.

† *Journ. Phys.*, vii and viii.

‡ *The Comparative Pathology of Inflammation*.

§ *Comptes Rendus*, tom. cxii, p. 842.

* *La Contratilité der Capillari*, 1881.

† *Journ. Physiol.*, vol. x.

to explode when the blood is shed. Similarly, Burdach found that the dog, which is naturally immune to anthrax, becomes susceptible after the injection of a quantity of fine sterile powder, for now the phagocytes can absorb no more, and the bacilli remaining outside the cells can develop freely.

Ruffer has also shown very neatly that the chemiotactic influence existing between the leucocytes and bacillary products may be annulled by the simultaneous injection of their toxins, preventing the great migration of leucocytes into the anterior chamber of the eye which normally follows the introduction of a drop of a culture of *Bacillus pyocyaneus*. Or the irritability of the sporadic mesoblast may be lowered in other ways, as by anæsthetics. Massart and Bordet* found in the frog that the migration of leucocytes towards tubes of living or sterilised cultures of bacilli was greatly diminished when the animal was anæsthetised. According to them, anæsthetics facilitate or aggravate infection by suppressing the irritability of the phagocyte. I imagine that for this purpose the drug must be pushed to a narcotic or poisonous degree.

In this connection it may be noted that frogs, which are normally immune to anthrax, become susceptible on being warmed. Kanthack and Hardy found in this case that the cells became completely paralysed, and showed no movement for five hours.

In fact, everything which lowers the irritability of the sporadic mesoblast increases the susceptibility of the organism to foreign invasion.

As to the functions served by this irritability, two stand out pre-eminent—the arrest of hæmorrhage and the removal of matter harmful or useless to the organism; for Hofmeister's† contention that one of the main functions of leucocytes is the absorption of peptone from the intestine is definitely set aside by the criticisms of Heidenhain and the observations of Metschnikoff (op. cit., p. 124) and of Shore.‡ This by no means excludes other activities; recent physiology has impressed on us the close interdependence of the bodily tissues. Hardy and Wesbrook§ have quite lately described changes in distribution of intestinal wandering cells during starvation and digestion, which suggest that they play a part in maintaining the "normality" of the blood plasma; of these, inflammatory changes are but a gross exaggeration.

(i) *The arrest of hæmorrhage.* It is agreed by all writers on the clotting of blood, except Wooldridge, that the disintegration of the white blood-corpuscle plays an important part in the process. In the crayfish we have seen that the disintegration of the explosive cell and the clotting of blood go hand in hand. But among invertebrates coagulable blood is the exception—the Arthropoda being the only group in which fibrin formation occurs, and here it is exceedingly rapid. This is probably connected with the rigid unyielding cuticle possessed by Arthropods, which renders strong contraction round the injured spot impossible. In the majority of invertebrates this contraction of the part instantaneously follows the infliction of a wound, so that the amount of blood lost is often very slight.

But in addition the process of plasmodium formation greatly assists the arrest of hæmorrhage. Geddes|| was the first to show that the non-granular cells of invertebrate blood rapidly coalesce on shedding to form a plasmodium, which acts as an efficacious plug to further bleeding; and that this process normally takes the place of clotting among invertebrates. Its details we have already considered.

It is obvious that in aquatic animals the arrest of hæmorrhage must be promptly effected, as the medium in which they live tends to favour its continuance just as a bath at the temperature of the body would favour it in man.

The Echinoderms are less well provided in this matter, for though they possess rigid exo-skeletons there is no fibrin formation, and if a considerable portion of the skeleton be broken, they infallibly bleed to death.¶ For smaller wounds, however, the rapid formation of a plasmodial plug appears to operate satisfactorily.

(ii) *The removal of matter harmful or useless to the organism.*

We have seen that matter naturally inert, such as carmine particles or fine sterile sand, is rapidly ingested, and we know that in town dwellers the bronchial glands may be black with soot ingested by phagocytes and removed from the lungs.

Moreover, they will ingest other leucocytes which have been injured in the conflict with micro-organisms,** or by the action of a poison such

as urari, which is known to cause a degeneration of leucocytes accompanied by extraordinary modifications in their shape.

Further, they will ingest micro-organisms which have previously been rendered inert. The mechanism by which the micro-organisms are actually killed we will consider shortly.

What is the ultimate fate of these ingested matters? The inefficient leucocytes and dead micro-organisms appear to be digested by the cells—that digestion taking place in an alkaline or neutral medium (Metschnikoff). But as to the innutritious foreign matter they ingest? Among the vertebrates the common mechanism appears for them to be removed from the general economy, still surrounded by cells, stored in the lymphatic glands, even ultimately invested by fibrous tissue. But among several classes of the invertebrates, at least, we meet with an interesting mechanism by which the cells actually migrate to the exterior, carrying the foreign matter with them. The cell sacrifices itself for the organism of which it forms part, and I am unaware of any chemiotactic reason on which such altruism can be explained at present.

Durham* found that insoluble granules introduced into the body-cavity of the starfish undergo ingestion by wandering cells, which then become adherent to the cœmic epithelium of the dermal branchiæ. Next by amœboid movements they force their way through the branchial wall to the exterior, where they disintegrate. If the process is going on actively, one may see little clumps of colour at the summits of the branchiæ, due to this emigration. These cells are termed "out-wandering cells," and by their agency the organism is rid of foreign matter. Green oysters show another interesting example of the same process. Their colour is due to the ingestion of a coloured diatom—*Navicularia ostrea*. If the oysters be removed to tanks from which this diatom is absent, they soon lose their colour, and colourless oysters soon acquire the characteristic hue if brought into the presence of the diatom. Apparently the insoluble pigment passes into the blood when the diatoms are digested, and by its continued accumulation would form a harmful element in the blood. In accordance with their ordinary activities the wandering cells ingest the pigment grains, and then pass, loaded with pigment, to the gills and palps. Ray Lankester,† to whom we chiefly owe our knowledge of this subject, believed that the pigment when it arrived at the gills was excreted by a number of "secretion cells" embedded in the superficial epithelium, these cells being green while the rest of the filament was colourless. He believed this in face of his own observation that these "secretion cells" could be noticed in a free amœboid condition crawling over the surface of the gill. Of course this at once suggests that the "secretion cells" are nothing more nor less than wandering cells pushing their way to the exterior. And lately Pelseneer‡ has traced the whole process more thoroughly, and has seen the amœboid blood-corpuscles loaded with pigment pushing their way to the exterior.

In fact this process may be followed with any insoluble pigment. While working at this subject I repeatedly had occasion to observe in Anodonta and Unio that within twenty hours of injection of carmine into the foot, a copious number of out-wandering cells were to be found on the gills, palps, and mantle, each cell loaded with carmine. At the same time active phagocytosis was progressing in the foot, the blood from the heart showed many beautiful examples of ingested particles, and the lacunar spaces of the gills, &c., were loaded with carmine containing cells pressing against the epithelium. All the stages in the history of an out-wandering cell could be traced.

Another point which seemed to be of interest, and which I have never seen referred to, was that the next nacreous layer of the shell to be formed was distinctly pink in colour, and on microscopical examination was found to be impregnated with carmine. Now, from the frequency with which excretory substances have been found in cuticular structures, it is believed by some that cuticle has a definite excretory value. I must not detain you longer on this point, but will refer § you to the discussion by Harmer in his paper on "Excretory Processes in Polyzoa." It seems to me, however, that the formation of a pigmented shell-layer during the active excretion of pigment is an additional fact pointing in the same direction.

Still more lately Lim Boon Keng|| has described the cells of the cœmic fluid in the earthworm out-wandering through the dorsal pores, and showing active phagocytosis in response to stimuli. And among the vertebrates Stöhr has found that the tonsils and Peyer's patches are sites for constant out-wandering. We do not know,

* Ann. Inst. Pasteur, tom. v, p. 417.

† Arch. f. exp. Path. und Pharm., Bd. xxiii.

‡ Journ. Physiol., vol. xi.

§ Ibid., vol. xviii.

|| Proc. Roy. Soc., 1880.

¶ Cuénot, Arch. Zool. Exp., 2nd series, tom. ix.

** Hardy and Lim Boon Keng, Journ. Physiol., vol. xv.

* Quart. Journ. Micr. Sci., vol. xxxiii.

† Ray Lankester, Quart. Journ. Micr. Sci., vol. xxvi.

‡ Ann. Soc. Roy. Malacol. de Belgique, tom. xxvii, p. 62.

§ Quart. Journ. Micr. Sci., vol. xxxiii.

|| Phil. Trans., vol. clxxvi, p. 122, 1895.

although it seems plausible, whether these cells remove noxious products also. At any rate Ruffer has observed that some emigrated leucocytes in the alimentary canal contain more or less digested microbes.

Taking the simpler cases among the invertebrates, it is noticeable that the cells which wander towards free surfaces usually contain pigment. Why they should out-wander we cannot say; it may be due in part to the stimulus afforded by the contents of the cell, in part due to light, possibly by the absorption of light by the pigment. But in this as in many other matters "the time is not yet ripe for dogmatism."

IV. Specialisation of the Structure and Functions of Sporadic Mesoblast.

We have hitherto spoken of the sporadic mesoblast as granular or non-granular. We have now to see that this corresponds with a very important difference in function. Just as the cells of other tissues have undergone differentiation in the more complex forms of life, so have the cells of the sporadic mesoblast.

The first to notice that some white blood-corpuscles were granular and others not, was Wharton Jones in 1846. Max Schultze in 1865 carried the analysis further in describing four types:—(a) Small round cell, non-amœboid, little clear protoplasm. (b) Larger cell, amœboid, more clear protoplasm. (c) Cells with finely granular protoplasm. (d) Cells with coarse granules in the protoplasm.

In a series of papers extending from 1878 to 1887, Ehrlich made a great advance by determining that these granulations had different specific reactions with aniline dyes, some having an affinity for acid dyes such as eosin, and others for alkaline dyes; some for both. The first were termed eosinophile granules, the second basophile, and the last amphophile. He also described neutrophile granules, but these have since been found by Kanthack and Hardy to really show an affinity for acid dyes, though less markedly than the first set.

The ordinary coarsely granular cells take the eosin stain to their granules with great avidity, while the most abundant leucocytes are "neutrophile" in his classification. But there is also in the blood a finely granular basophile cell of which the characteristic reaction is a rose colour stain with methylene blue, only properly seen by artificial light. Certain other plasma cells of the connective tissue, which he termed Mastzellen, have coarse granulations, also basophilic in reaction.

I will not deal with his work in greater detail, valuable pioneering though it was, because he did not attempt to determine the functions of the cell, of which we have more particularly to treat, and because his classification bids fair to be completely superseded by that of Kanthack and Hardy, which is at once simpler, more comprehensive, and more accurate.

The first suggestion as to the function of the eosinophile cells was due to Hankin,* of this hospital, who isolated *in vitro* a proteid substance from lymphatic glands which had a destructive action on bacteria. To such substances he believed the resistance of animals towards the growth of microbes to be due, and so he termed them "defensive proteids;" subsequently the name "alexines" was adopted. He associated the bactericidal powers of the body with the wandering cells by showing, in conjunction with Kanthack,† that "outside the animal body during fever a rise in the bactericidal power of the blood occurs *pari passu* with the increase of the number of leucocytes present." He then linked this increased bactericidal power of the blood with the eosinophile, or, to adopt the better and more convenient term, oxyphile granules, by showing that it is apparently associated with the discharge of these granules, coarse or fine, into the blood-plasma.

Hankin's ingenious theories of the conflict between the organism and the microbe have not all stood the test of time, but his suggestion that the oxyphile granules of the sporadic mesoblast have a bactericidal function has recently received support from the admirable researches of Kanthack and Hardy,‡ to which we will now turn. They started from the observation that the leucocytosis induced by the injection of pathogenic microbes was different in character from that following injection of inert matter, such as finely divided coagulated proteid or Indian ink. While the latter is followed by a large increase of the non-granular phagocytes, or, as they termed them, "hyaline" cells, it is the oxyphile cells, especially those with coarse granulations, that accumulate when pathogenic microbes are introduced.

Now the frog is normally immune to anthrax; the stages in the

conflict can be watched in a hanging drop of lymph under the microscope, the same cells being followed throughout.

Stage i.—The first cells to enter the field of action are the oxyphile cells, which, strongly attracted towards anthrax, come into contact with the chains of bacilli. The oxyphile cell is ordinarily very sluggish, but under these circumstances it is profoundly stimulated, and exhibits quick streaming movements of the cell substance; and then the oxyphile granules are discharged, those nearest the bacillus fading and dissolving first. The victory at this stage appears to rest with the army numerically greatest; if the oxyphile cells are unharmed by the bacilli, they bud off daughter-cells at first free from granules. These creep away from the field of battle till their granules are formed, when they seek the same or another focus of conflict.

Towards the end of this stage the oxyphile cells form a plasmodium around the bacilli, though their endosare and granules remain distinct. The bacillus is only injured near the oxyphile cell, where the contents become rapidly curdled and irregular, or may be completely dissolved; but the mere vicinity of this cell may profoundly arrest its development. It may be noted that the reaction by greatest area of contact still holds here, for if the oxyphile cells be in the minority they will extend themselves to the most attenuated lengths in order to attack as great a length of the bacillary chain as possible.

While all these manoeuvres are progressing the phagocytes or hyaline cells are not attracted towards the bacilli, though they can still take up indifferent matter, such as Indian ink; but in the vicinity of an active bacillus they seem to be paralysed.

Stage ii.—The hyaline cells have now increased in number, and fuse with the oxyphile cell surrounding a bacillus, at the same time rolling the long-drawn-out mass of cells and bacilli up into a ball. This fusion into a lobed plasmodium may last for one or two hours.

Stage iii.—The cells begin to shed off and regain their individuality. We then find that during the second stage a manoeuvre has occurred which may be compared to the "ladies to the centre" movement in the third figure of the Lancers—that is, the hyaline cells, originally outside, have taken up their position in the centre of the plasmodium, while the oxyphile cells are now at the periphery, and are the first to separate off. The central hyaline mass is now seen to contain the bacillary remains enclosed in food-vacuoles.

Stage iv.—The hyaline mass now separates into its original constituent elements. By this time the basophile rose-colouring cells have increased in number and also in size, so that their cell-substance becomes completely filled with granules showing the characteristic reaction. Kanthack and Hardy sum up the conflict as follows:

1. The maiming of the bacilli by the oxyphile cells.
2. The removal of the remains of the bacilli by means of the ingestive and digestive activity of the hyaline cells.
3. The probable removal of dissolved foreign substances by the rose-staining cells. The accumulation of bacterial toxins beyond a certain point paralyses the oxyphile cells and destroys the hyaline. They suggest that the increase of the amount of rose-staining substance is correlated with the removal of bacterial products.

Hardy and Keng* followed the subsequent fate of the oxyphile cells that had taken part in the conflict, and found that though they apparently were loaded with normal granules, they were below that standard of efficiency demanded by the body, and were in consequence attacked and ingested by the hyaline cells. Maimed and weakened cells are, therefore, prevented by this cannibalism from again entering the conflict. It is highly probable that all the descriptions of supposed phagocytosis by oxyphile cells are really instances of hyaline cells containing remains of such oxyphile cells.

More recently Kanthack and Hardy have extended their results to Mammalia, and have classified the granular cells into—

I. Oxyphile: (a) Finely granular ("neutrophile" of Ehrlich), typically found in the blood. (b) Coarsely granular, typically found in the coelomic and lymphatic spaces.

II. Basophile: (a) Finely granular, in the blood. (b) Coarsely granular, in connective-tissue spaces ("explosive" in guinea-pig and rabbit).

Thus we find they can be divided into a hæmal group—typically finely granular—and into a group found in the coelomic and interstitial spaces, typically coarsely granular. An irritant near the blood-vessels of a vascular membrane will provoke a reaction of cells of the hæmal type; while a Ziegler chamber or capillary tube, filled with bacilli or their products, introduced into the peritoneal cavity induces a reaction solely of the coelomic cells. But under all such circumstances the coarsely granular oxyphile cell, the eosinophile cell *par excellence*, is the first to enter the field of conflict, and to suffer loss of granulation, while phagocytosis follows, being accomplished by the hyaline cells.

* Hankin, *Brit. Med. Journ.*, July, 1890; *Centralblatt f. Bakt. u. Par.*, 1892-3.

† *Proc. Camb. Phil. Soc.*, vol. vii.

‡ *Phil. Trans.*, 1893.

* *Loc. cit.*

In connection with the solvent action of the oxyphile granules it is noteworthy that a small strip of copper introduced into the anterior chamber of the eye is dissolved, and that of the cells present during the process 95 per cent. are coarsely granular oxyphile cells.

The proof of the different functions of these cells has by Kanthack and Hardy's last paper been made well-nigh complete. We find, in fact, that the method of extra-cellular digestion, found among comparatively primitive forms of life to be the most effectual means for nutritive purposes, has even been adopted by the sporadic mesoblast, the last remnant faithful to the old intra-cellular method.

We find, for instance, "within the limits of a single group of animals that the simplest forms possess only one kind of wandering cell, while those of greater structural complexity have all three typical forms. Thus the leucocyte in a primitive Arthropod like *Daphnia* is granular, protective, digestive, absorptive, and constructive (for it contributes to the formation of fat and scar tissue), and its granulation is amphophile and rose-staining." Now in the crayfish, a much more highly differentiated Arthropod, we find the three types of cells—oxyphile, basophile, and hyaline—differentiation is well marked. The oxyphile cell has accentuated the glandular and protective character of the primitive cell. Just so extra-cellular methods of slaying the prey obtain among the more specialised Protozoa, as *Vampyrella*. Both in the specialised Protozoan and the specialised leucocyte three stages are seen.

1. The contact with the prey stimulates the captor to excrete a poison.
2. The now inert body is ingested.

3. There is a secretion of a digestive fluid, which dissolves the ingested prey (Kanthack and Hardy). These last two stages are, of course, performed by the hyaline cell. Then the absorptive powers of the primitive cell are represented by the rose-staining cell of the more differentiated animal forms. The constructive function in higher forms is relegated to the connective-tissue cells.

It is interesting, further, to note that a foetal mammal has only one type of wandering cell, differentiation occurring subsequently, reminding us again, in Marshall's terse phrase, that every animal climbs up its own genealogical tree.

Hence we may say that the granular cells have adopted the more specialised method of extra-cellular digestion, while the hyaline cells retain intracellular digestion as their method. And these processes exist side by side, just as they do, for instance, in the intestinal digestion of the earthworm, where, as Miss Greenwood* has shown, ingestive cells with retractile cilia and digestive cells with granulations are both to be found.

Metschnikoff has adopted a much narrower view of the process, and,

in referring all questions of resistance and immunity to phagocytosis, has lost sight of this more general and more potent method of extra-cellular digestion.

All the recent advances in serum-therapy, however, point in the direction of this extra-cellular process. Pfeiffer has recently noted that under certain circumstances, when guinea-pigs had been rendered highly refractory to the spirillum of cholera, these microbes became swollen and spherical on injection into the peritoneal cavity before any phagocytosis had time to come into play. Here, then, is another example of extra-cellular digestion by mesoblast.

It is true that instances of the ingestion of living microbes have been witnessed. The *Vibrio Metschnikovi*, if inoculated into the anterior chamber of the eye, are soon included in phagocytosis. And these phagocytes, if placed in broth, perish, while the vibrios emerge and develop, apparently none the worse for their temporary interment. Nevertheless, speaking generally, it appears that the more virulent the microbe, the less the proportion of them taken up by the cells, and the longer time the phagocytes take to come into action. In these cases extra-cellular destruction is usually a preliminary measure.

The view of this differentiation of functions in the sporadic mesoblast has been attacked from another standpoint, and it is urged that these cells are simply varieties of a single type. Martin Heidenhain has urged that the coarsely granular oxyphile cell is an over-ripe leucocyte undergoing degeneration, which is highly improbable in face of its robust reactions and high resistance.* Gulland† has pointed out that oxyphile material is abundant in foetal lymphatic glands where there is scant need for protection from microbial invasion and great need for nutrition. He agrees with Metschnikoff that they probably represent reserve material resembling yolk or aleuron grains, which are also eosinophile. Whether all oxyphile material is one and the same I should hesitate to say, but I cannot consider the fact that eosinophile cells are abundant in the foetus sets aside the many examples in which Kanthack and Hardy have followed the reaction of these cells to microbes step by step.

And against these varieties of sporadic mesoblast being simply pleomorphism of a single type we may urge (a) the existence of immature examples of the different forms; (b) the fact that in highly differentiated animals the same cell never contains at one and the same time two different kinds of granules (Sherrington).

Moreover it would be strange were the sporadic mesoblast exempt from that increasing differentiation which is true for all other tissues.

* See Sherrington, *Science Progress*, February, 1895.

† Gulland, Reports from Lab. of R.C.P.Edin., vol. iii, *Journ. of Pathology*, May, 1894.

CLASSIFICATIONS OF THE CELLS OF THE SPORADIC MESOBLAST.

Wharton Jones, 1846.	Schultze, 1865.	Ehrlich and pupils, 1878-87.	Metschnikoff, 1892.	Kanthack and Hardy, 1894.
Nucleated cells	(i) Small; non-amœboid (ii) Large; amœboid	(i) Lymphocyte (ii) Large mononuclear cell	(i) Lymphocyte (ii) Mononuclear leucocyte	(i) Lymphocyte. (ii) Hyaline cell.
Granule cells (i) Finely granular	Granular cells (iii) Finely granular	(iii) Neutrophile cell— ϵ (amphophile in rabbits and guinea-pigs) (iv) Eosinophile cell— α	(iii) Polynuclear leucocyte (neutrophile) (iv) Eosinophile leucocyte	(iii) Oxyphile cells. (a) Finely granular (hæmal). (β) Coarsely granular.
(ii) Coarsely granular	(iv) Coarsely granular	(v) Basophile cell— δ (vi) Mastzellen (connective tissue). Coarsely granular basophile cell— γ		(iv) Basophile cells. (a) Finely granular (hæmal). (β) Coarsely granular.

May I briefly recall the somewhat devious steps of our argument? We have attempted to trace the genealogy of the phagocyte, and have seen how it retains many ancestral traits; we have seen that it reacts to various stimuli, being attracted by certain substances and repelled by others; that in presence of foreign matter it offers the largest surface of contact possible, and (a fact often overlooked) that the intensity of the reaction depends closely on the stability of the cell as well as on the intensity of the stimulus. Further, we saw that this reaction is not limited by the body-walls, for a cell may actually wander outside the body in obedience to stimulation. We found that the wandering cells have undergone differentiation like other tissues, and that in the higher forms phagocytosis does not include the whole of the mechanism at the disposal of the organism against microbial invasion. And we have briefly summarised the functions subserved to the organism by the irritability of these cells.

Perhaps the chief idea which a study of these mechanisms leaves with us is the extraordinary complexity of protoplasmic reactions, a complexity behind which lies the mystery of life, a mystery as little fathomed to-day as in the first grey dawn of scientific research.

Smoking Concert Club.

THE third Smoker of the season was held on February 1st, at the Frascati Restaurant, but, owing to a series of unfortunate accidents, was not equal to either of its predecessors. That indispensable official, the pianist, was disabled by an accident to his finger, and his substitute never discovered the whereabouts of the concert! Half an hour after the advertised time for the overture, no musician having turned up, things were getting desperate, and a "new growth," in the shape of a "hump," appeared on the Chairman, the Secretary, and the forty or fifty gentlemen who formed the audience. The Secretary to the rescue, and proceedings began with a "tune" on the piano. Then followed various songs by Messrs. S. F. Smith, C. G. Meade, J. Macauley, Dick Welch, J. K. Birdseye, and W. Long. These gentlemen spared themselves no pains to entertain, and were certainly very successful in reducing the "hump," though of course much handicapped by the absence of the professional pianist. Mr. J. C. Powell, the other Hon. Sec., spent the major part of the evening in a cab looking for an "ivory-scratcher," apparently a much rarer bird than the "wood pecker," and not to be caught on Saturday nights; but being unsuccessful his colleague filled the gap as best he might. In the course of the evening the Chairman, Mr. D. L. E. Bolton, said that he deplored the poor support the efforts of the executive had received on this occasion, and the apparent want of *esprit de corps* amongst members of the Club; he hoped that in the future the concerts would be given before "bumper" houses.

Dr. Haydon, who is always ready to give us the benefit of his services, played two violin solos—Raff's "Cavatina," and Simonetti's "Madrigale"—in his finished and artistic style. Mr. S. F. Smith played several banjo solos, filling in the gap made by Mr. Stanley Gibson's absence most efficiently; these, his first efforts in this line that the Club has had the pleasure of hearing, were received with great applause. Mr. Macauley was a host in himself, and sang and recited admirably again and again. Messrs. C. G. Meade, W. Long, and Dick Welch supplied the comic element, and sang all the well-known comics of the day in their various styles, but with equal success. Shortly after eleven the concert concluded with "Auld Lang Syne" and "God save the Queen."

At a committee meeting held in January, Mr. P. W. James was elected vice-chairman, vice Mr. F. W. Gale resigned. Mr. F. E. Meade was elected to fill Mr. James's place on the committee.

Cases of Special Interest.

Medical.

- Matthew, bed 1.—Pseudo-hypertrophic paralysis.
- Matthew, bed 10.—Hodgkin's disease.
- Matthew, bed 11.—Spasmodic torticollis.
- Luke, bed 13.—Optic neuritis.
- Colston, bed 19.—New growth of liver.
- Colston, bed 14.—Chronic jaundice.
- Mark, bed 21.—Double aortic disease in a boy.
- Faith, bed 2.—Alcoholic neuritis.
- Hope, bed 11.—Congenital spastic paraplegia.
- John, bed 17.—Syphilitic cachexia.
- Mary, bed 17.—Typhoid fever.

Notes.

WE ARE glad to see that Mr. A. W. R. Cochrane, who passed second into the Indian Medical Service in August, has improved his position at Netley. He is at the head of the list with 100 marks to spare. Mr. R. P. Wilson, for some time a post-graduate student here, was fifth.

C. H. R. PENTREATH, having completed his examinations and read his thesis, has taken the degrees of M.B. and B.C. at Cambridge. L. B. Burnett has also taken the M.B. degree.

AT THE University of London, the question of the reconstruction of the University on the lines of the report of the Cowper Commission has again been before Convocation. This time the majority in favour of the Scheme was larger than ever, and the following resolution was carried by 466 votes against 240, viz. "That this House desires the early introduction into Parliament of a Bill for the reconstruction of the University similar to that introduced last year by Lord Playfair, but with an inserted clause securing to the Senate, to Convocation, and to other bodies affected, the right of appeal to the Privy Council, on any of the provisions which may hereafter be settled by the Statutory Commission."

DR. DONALD MACALISTER has been added to the Commission of Peace for the county of Cambridge.

J. W. HAINES, M.B., B.S., has been admitted a Fellow of the Royal College of Surgeons.

BART'S men who have recently started in general practice are—A. J. H. Boyton at Ilford; W. G. H. Bradford at Debenham, Suffolk; R. E. Crosse at Dereham, Norfolk; B. W. Gowing at Newport, Mon.; C. W. Grant at Portsmouth; P. Lambert at Newmarket; W. L. Pethybridge at Plymouth; G. P. Shuter at Chiswick; C. S. de Segundo in Bentinck Street; B. B. Thorne-Thorne at Woking; L. C. Thorne-Thorne at Inverness Terrace; B. P. Viret at Bradford; C. H. Whitford at Plymouth; N. O. Wilson at Dover.

MR. D'ARCY POWER gave a most interesting lecture on February 10th, at the London Institution, Finsbury Circus, on the "Meals of our Ancestors." His lecture included many historic details in the development of our present habits and manners at table, and the many changes in the time and arrangement of the various daily meals.

Correspondence.

To the Editor of St. Bartholomew's Hospital Journal.

SIR,—In connection with your note of warning last month, may I ask old Bart's men through your columns who have received any such letters to communicate with me? In doing so they will be doing a public service, and personally oblige—Yours, &c., F. J. DIXON.

BELLEVUE, HERNE HILL, LONDON;
January 29th, 1896.

Amalgamated Clubs.

BALANCE-SHEET, 1894-5.

Cr.	£	s.	d.	Dr.	£	s.	d.	£	s.	d.
By Members' Subscriptions	649	19	0	To Grants to Clubs :						
" Grant from Medical School	100	0	0	Rugby Football Club	55	11	3			
" Profit on the JOURNAL Account	159	5	1	Association Football Club	39	0	11			
				Boxing Club	21	11	10			
				Athletic Club	56	2	3			
				Cricket Club	20	15	11			
				Swimming Club	11	15	4			
				Lawn Tennis Club	3	3	0			
				Boating Club	3	17	2			
								211	17	8
				To Abernethian Society, 107 members at £1 1s. ...				112	7	0
				" Musical Society				20	0	0
				" Maintenance and Reserve Fund				564	19	5
								£909	4	1

Audited and found correct according to
vouchers and bank pass book.

PERCY FURNIVALL.
H. MORLEY FLETCHER.
W. REGINALD STOWE.

£909 4 1

January, 1896.

MAINTENANCE AND RESERVE FUND, 1894-5.

Cr.	£	s.	d.	Dr.	£	s.	d.
By Balance from 1893-4	205	14	1	To Stamps for cheques	0	8	4
" Funds as per General Account	564	19	5	" Subscriptions to Hare and Hounds	3	3	0
" Sale of Refreshments	15	0	0	" Special Grants to Clubs :			
" Fines for new Tickets	0	3	0	Rugby and Association Football Clubs	5	0	0
				Swimming Club	3	13	0
				" Furnishing pavilion	120	4	8
				" Rollers and appliances for ground	116	9	7
				" Rent	75	0	0
				" Rates, taxes, and water	30	6	0
				" Coal	4	19	0
				" Seeds and turf	10	3	6
				" Flag and flagstaff	8	1	9
				" Keys for lockers	7	10	0
				" Horse	10	0	0
				" Printing	1	2	6
				" Wages of clerk	5	0	0
				" Wages of groundmen and boy and sundries	107	8	1
				" Refreshments, luncheons to visiting teams, &c.	19	10	6
				" Secretary's petty cash	7	0	0
					£534	19	11
				" Balance to next account	250	16	7
					£785	16	6

Audited and found correct according to
vouchers and bank pass book.

PERCY FURNIVALL.
H. MORLEY FLETCHER.
W. REGINALD STOWE.

£785 16 6

RUGBY FOOTBALL CLUB.

BART'S v. WICKHAM PARK.

Played at Catford on January 11th, and resulted in a draw of 3 points all. The game throughout was of a scrambling and uninteresting nature, there being an entire lack of dash and combination. The ground was in a heavy and slippery condition.

In the first half the game was very even, play being almost entirely confined to the forwards. Just before half-time Bennett injured his shoulder, and was practically useless for the rest of the game.

In the second half Wickham Park had rather more of the play, but were never really dangerous. The Hospital was penalised for off-side play, and from the kick Wickham Park scored a goal. Immediately afterwards Robbs dribbled up from halfway and scored a good try, but the kick went wide. Just on the call of time Body ran right through to the back, and had he passed a certain try must have resulted.

Team.—H. Bond (back); A. J. W. Wells, T. M. Body, A. E. Hodgkins, S. Mason (three-quarters); A. Hawkins, G. C. Marrack (halves); J. C. A. Rigby, H. M. Cruddas, W. F. Bennett, J. K. S. Fleming, C. H. D. Robbs, W. M. James, F. G. Richards, A. C. Adams (forwards).

ST. BART'S v. LEICESTER.

Played at Leicester on Monday, January 20th, and resulted in a crushing defeat for the Hospital by 7 goals and 3 tries to nil.

Bart's were very weakly represented, as Bond, Marrack, Wells, Body, Hodgkins, and Bennett were absent. No description is needed, as Leicester did practically as they pleased.

Team.—S. F. Smith (back); S. Mason, C. Dix, W. H. Randolph, F. J. Wood (three-quarter backs); A. Hawkins, T. Martin (half-backs); P. O. Andrew, J. C. A. Rigby, H. M. Cruddas, J. K. S. Fleming, C. H. D. Robbs, W. M. James, H. C. Adams, H. Weeks (forwards).

BART'S v. NORTHAMPTON.

This match was played at Northampton on January 25th, and resulted in a win for the home team by 2 goals and 1 try to a dropped goal and try, or by 13 points to 7. The Hospital was again weakly represented.

Within two minutes of the start one of the opposing three-quarters intercepted a pass, and running the whole length of the field scored a try under the posts, which was converted. On starting again the Hospital forwards began to press, but could not get through for some time. Finally Robbs got over, but the kick failed. Almost immediately afterwards Hawkins picked up the ball from a scrum in front of goal, and dropped a goal. Nothing more was scored up to half-time.

From the kick off the Northampton forwards rushed the ball over and scored a try, which was not improved upon. For the whole of the remainder of the game the Hospital forwards were on the Northampton goal line, and had very hard luck in not scoring. Just on the call of time an opposing three-quarter intercepted a pass and scored.

Team.—H. Bond (back); S. Mason, W. H. Randolph, A. E. Hodgkins, and T. M. Body (three-quarter backs); A. Hawkins, G. C. Marrack (half-backs); H. M. Cruddas, J. K. S. Fleming, C. H. D.

Robbs, W. M. James, F. J. Wood, H. Weeks, A. L. Vaughan, and A. N. Other (forwards).

ASSOCIATION FOOTBALL CLUB.

Saturday, Jan. 11th.—ST. BARTHOLOMEW'S HOSPITAL *v.* HARROW ATHLETICS.

This match was played at Harrow before a good many spectators, and resulted in the defeat of the Hospital by two goals to none. The Hospital were perhaps handicapped by the ground, which was, to say the least of it, unfamiliar, and also by the absence of Pickering, whose place, however, was very ably filled by Woolcombe.

Harrow started, and immediately gained Bart.'s territory, the ball, however, being cleverly obtained by Joy, who passing Willett took the ball well up only to shoot behind. Harrow again got possession after desultory play in the centre, and forcing the ball down, the centre forward put the ball into the net. Again before changing ends the same player shot, and the ball rebounding off Fox was headed into the net. Thus in spite of several runs by Hay, and good, bad, and indifferent shots from our forwards, Harrow led by 2—0. On changing ends things looked better for Bart.'s, who constantly besieged the Athletic goal, and the excellence of their goal-keeper and our own bad luck only preventing us scoring several times, shots grazed the bar only to go behind or to end in corners. It is not too much to say that after half time Bart.'s had absolutely the best of the game. This has happened this year several times. Why cannot Bart.'s get goals? Certainly in this match lack of energy among the inside forwards was the sole reason. Had Willett used more of his weight with the goal-keeper the result would have been different. As it is, Bart.'s has to make what it can of a defeat by Harrow Athletic of two goals to none.

Team.—E. H. B. Fox, goal; R. P. Brown and L. E. Whitaker, backs; A. H. Bostock, N. H. Joy, A. Woolcombe, half-backs; T. H. Talbot, R. Waterhouse, J. A. Willett, E. W. Woodbridge, and A. Hay, forwards.

Monday, Jan. 13th.—ST. BARTHOLOMEW'S HOSPITAL *v.* OLD REPTONIANS.

This match was played at Winchmore Hill. The Old Reptonians kicked off from the pavilion end, and started what proved to be a very keen and evenly contested game. No points were scored in the first half, though both goals were frequently attacked, more especially that of Bart.'s, whose goal-keeper Fox was, however, quite equal to the emergency, saving time after time in grand style. After changing ends play became more exciting; after a few minutes' play Bryant scored the first goal for the Old Reptonians by an excellent shot. By no means dismayed, Bart.'s kept the ball as much as possible in the opponents' half till Talbot by a pass from Wethered put the ball into the net, thus making the score stand 1—1.

This brought the play to within about a quarter of an hour of time, and the keenness of the play continued unabated. The Reptonian forwards, though passing well, were constantly being brought to a standstill by the excellent tacking of Bostock, while on the left Tweedie did excellent work. Finally, from a *mêlée* in front of the Bart.'s goal the ball was sent out to Hay, who taking possession, ran the whole way down the ground, and out-distancing his opponents, put the ball into the net, thus putting to Bart.'s credit the winning goal, thus 2—1. Shortly after this time was called.

Considering there were only four of the usual team playing, Bart.'s may be congratulated on making a thoroughly successful victory of what, on paper, looked like a certain defeat.

Team.—E. H. B. Fox, goal; C. G. Watson and A. H. Hayes, backs; A. Woolcombe, A. H. Bostock, and A. R. Tweedie, half-backs; A. Hay, J. D. Hartley, E. Wethered, E. G. Simmonds, and T. H. Talbot, forwards.

Wednesday, Jan. 15th.—ST. BARTHOLOMEW'S HOSPITAL *v.* GRAVESEND UNITED.

This match was played at Gravesend before a large number of spectators, and resulted in a win for Gravesend by nine goals to nil, a result not surprising considering the Hospital were minus the services of Messrs. Willett, Robinson, Pickering, Joy, and Fox, and that during the match Brown was replaced by Watson owing to injury, and Talbot was also disabled.

There was an immense wind blowing down the ground, and Bart.'s elected to play with it first half. Gravesend scored twice during this portion of the game. On crossing over, play became of a very one-sided nature, and though Bart.'s made several commendable attempts to force the ball away their efforts were for the most part useless. Gravesend, sending in shot after shot, scored seven times, the scores at the end standing nine to nil.

Team.—A. Pugh, goal; C. G. Watson and L. E. Whitaker, backs; J. C. Marshall, A. H. Bostock, and A. R. Tweedie, half-backs; T. H. Talbot, C. Grimshaw, R. Waterhouse, E. W. Woodbridge, and A. Hay, forwards.

LONDON CUP TIE.

Saturday, Jan. 25th.—ST. BARTHOLOMEW'S HOSPITAL *v.* OLYMPIANS.

This Cup Tie, in the first round of the London Senior Cup, was played at the Olympian ground, Walthamstow, in the presence of a large crowd of spectators. The result was Bart.'s 2, Olympians 1. The ground was in a very muddy condition, and the weather was not very favorable; however, the game resulted in some very fair play on both sides. The Hospital were without the services of Messrs. Robinson and Joy, their respective places being taken by R. Waterhouse and A. Woolcombe.

Bart.'s won the toss and decided to play uphill with the little wind there was blowing. The Olympians then kicked off, and their forwards, after some clever passing, were pulled up by Bostock, who passing to Prance, the latter by the aid of Hay took the ball up the field, —the shot, however, going behind. The ball was for some minutes kept close in the Olympian quarters, shots being sent in by the Bart.'s forwards, —most, however, going wide. The Olympians then woke up, and Blundell getting possession, in concert with the inside right brought the ball down the field, and through an error in judgment between the Bart.'s backs and goal-keeper, the latter of whom ran out unadvisedly, put the ball into the net. The score now read Olympians one, Bart.'s nil.

The ball was then kicked off, and the Bart.'s forwards playing well and hard took the ball up, and Woodbridge, from a pass by Waterhouse, by a splendid shot which few goal-keepers could have stopped, scored a point for the Hospital, thus making the score 1—1. Play now ruled very fast, and the backs of both teams were kept well employed, and Bart.'s after pressing for several minutes scored another point by Waterhouse from a good shot. Scores at half-time were two to one in Bart.'s favour.

On crossing over, Bart.'s, playing downhill, had a good deal of the best of the game, and except on one or two occasions Fox was hardly requisitioned at all. The Hospital, however, continually looked dangerous, Hay especially running several times down the ground, only just failing to score. Shots were constantly being sent in, and Waterhouse from a pass by Woodbridge sent in a beautiful low shot which was just saved by the Olympians' custodian. After this play ruled more even, and no points were scored till the whistle blew, leaving Bart.'s victorious by two goals to one.

The Hospital may be congratulated on their victory, both because they had not played for some time before, and because of the absence of two of their men.

The goal that was scored against them was one that Fox would easily have stopped had he not been called out by the backs, and so was unable to get back in time. Prance playing in Woodbridge's usual place played up very hard, and did some very useful work, as did Woolcombe at half-back.

Team.—E. H. B. Fox, goal; R. F. Brown and L. E. Whitaker, backs; A. Woolcombe, A. H. Bostock, and H. J. Pickering, half-backs; T. H. Talbot, R. Waterhouse, E. W. Woodbridge, C. H. G. Prance, and A. Hay, forwards.

Referee—Lieutenant Chase (A.S.C.).

We regret to hear that J. A. Willett has been advised by his doctors not to play football any more this season, so he will not be able to take his place as centre forward in the forthcoming Cup ties.

DRAWS FOR THE UNITED HOSPITAL ASSOCIATION CUP.

First Round.—To be played on or before January 31st.

A. St. Bartholomew's *v.* King's.

Second Round.—To be played on or before February 14th.

B. Winner of A. *v.* Charing Cross.

C. London *v.* St. Thomas's.

D. Middlesex *v.* University.

E. Guy's *v.* St. Mary's.

Semi-Final on or before February 28th.

F. Winner of D. *v.* Winner of E.

G. Winner of C. *v.* winner of B.

Final.

H. Winner of F. *v.* winner of G.

The semi-final and the final are to be played at Leyton.

First named to have choice of ground in first and second rounds.

In the first round which was to be played on January 30th, King's Hospital scratched to St. Bartholomew's, as they were unable to raise a team.

Abernethian Society.*

N November 14th, Mr. Langdon Brown read a paper on "The Mechanism of Phagocytosis," the President, Mr. Murphy, in the chair. Mr. Brown's paper was a well arranged and thorough monograph, which gave his hearers a clear idea of a subject which is practically a "dark continent" to most of us.

On November 21st, Mr. J. Hussey read a paper on "The Blood Parasites in Malaria," the President, Mr. Meakin, in the chair. This paper, which was largely based on Mr. Hussey's original work, also laid a considerable amount of information before the Society, on a subject which is barely treated in the text-books. The reader illustrated his paper with excellent diagrams and microscopic specimens; the latter were particularly good.

On November 28th, Mr. Paterson read a paper on "Some Points in the Diagnosis of Typhoid Fever," the President, Mr. Murphy, in the chair. A long discussion followed, the merits of the Diazo reaction were criticised, and the forest of symptoms which have occurred in various cases were borne witness to, showing the extreme difficulty there may be in making a diagnosis.

On December 15th, the President, Mr. H. B. Meakin, being in the chair, a paper on "Photomicrography" was read by Mr. E. W. Roughton in conjunction with Mr. C. H. Cosens. Over 100 members attended this meeting, which was held in the Anatomical Theatre. The paper was prefaced by an explanation of the various apparatus employed, which had been brought and set up with considerable trouble by Messrs. Roughton and Cosens. The photographs were a revelation to the great majority of those present, and are probably unique in their excellence. A large series were shown. Dr. Kanthack, who had prepared the specimens from which the photographs were taken, said he hoped in time to come to illustrate his pathological lectures with photomicrographs. In the course of the evening he showed a photograph of a drawing by Mr. Smith of actinomycosis executed thirty years ago, when the fungus had not been recognised.

This paper brought the first half of the Session to a close.

Volunteer Medical Staff Corps.

NO. 3 (LONDON) COMPANY.

THIS Company is composed of students of St. Bartholomew's and St. Thomas's Hospitals only. The officers are Surg.-Capt. H. Work Dodd (officer commanding), Surg.-Capt. G. Sims Woodhead, Surg.-Lieut. H. J. Waring (company adjutant).

Any gentleman desirous of joining should do so at once. All particulars can be obtained from either of the following N.C.O.'s: Staff-Sergts. H. G. McKinney and J. C. S. Dunn, Sergts. T. Compton and A. Granville.

The Bart's half-company drill every Tuesday in Charterhouse Square at 4.30 sharp.

The following Corps parades are down for February:—Saturday, Feb. 15th.—Chelsea Barracks, 4.30 p.m., dress, drill order with leggings and rolled great-coats; band to attend. Saturday, Feb. 29th.—Fall in at Finchley Road Station at 4.45 p.m., dress as above; band to attend.

Examinations.

FIRST CONJOINT: *Chemistry and Physics*.—T. M. Body, H. E. Flint, C. S. Hawes, G. J. A. Leclizio, C. C. B. Thompson, P. L. Vawdrey, C. C. K. White, and N. Walmisley.

FIRST CONJOINT: *Pharmacy and Materia Medica*.—H. G. Harris, L. Jones, W. T. Storrs, G. P. Taylor, L. L. Allen, F. Harvey, and A. A. Humphreys.

FIRST CONJOINT: *Biology*.—C. Fisher and G. S. A. S. Wynne.

FINAL L.S.A.: *Forensic Medicine and Midwifery*.—H. C. Wimble.

FINAL L.S.A.: *Surgery, Medicine, Forensic Medicine, and Midwifery*.—W. Hampson.

FINAL CONJOINT.—The following having passed in all subjects of the final examination have received their Diplomas of M.R.C.S. and

* Received too late for publication in the January number.—Ed.

L.R.C.P.:—G. A. Anden, H. J. Bumsted, W. J. Codrington, M. W. Coleman, W. F. Cross, S. J. O. Dickens, A. E. Druiett, J. Evans, J. C. Fisher, J. M. Flavell, A. Heath, J. Hobday, J. Hussey, H. F. Hyde, P. T. Jones, E. D. M'Dougal, J. P. Maxwell, H. J. May, H. B. Milsome, J. C. Padwick, R. D. Parker, T. S. Pigg, W. H. Pope, A. R. H. Skey, J. Thomas, C. H. Wilmer and R. W. Jameson.

PRIMARY L.S.A., PART II: *Anatomy and Physiology*.—L. T. Lavan, E. B. Stevenson. *Anatomy*: H. J. Pickering. *Physiology*: P. Cator.

SECOND CONJOINT: *Anatomy and Physiology*.—P. C. Barham, E. P. H. Dudley, A. G. Higgins, J. G. F. Hosken, H. P. Lobb, A. L. Scott, H. E. Waller. *Anatomy*: G. B. Nicholson, C. D. Parfitt, J. E. Robinson. *Physiology*: A. Hawkins, C. D. Parfitt, J. E. Robinson.

Appointments.

SNOW, L. U., M.R.C.S., L.R.C.P., has been appointed Medical Officer and Public Vaccinator for the No. 2 District of the Horsham Union.

BACK, A. H., M.A., M.R.C.S., L.R.C.P., has been appointed Medical Officer and Public Vaccinator for the fifth District of the Aylsham Union.

BACK, H. H., M.B.(Lond.), M.R.C.S., has been appointed Medical Officer of Health to the Aylsham District Council.

BARKER, TOFT, M.R.C.S., L.R.C.P., has been appointed House Surgeon to the Victoria Hospital for Children.

THORNE-THORNE, LESLIE, M.D.Dur., has been appointed Clinical Assistant to the Paddington Green Children's Hospital.

FOULERTON, ALEX. G. R., F.R.C.S.Eng., D.P.H.Camb., has been appointed Demonstrator of Biological Chemistry to the British Institute of Preventive Medicine.

CLAPHAM, Surgeon-Captain, A.M.S., has been posted to Colchester.

Pathological Department of the Journal.

SPECIMENS sent by subscribers to the JOURNAL will be examined in the Pathological Laboratory and a report furnished under the supervision of Dr. Kanthack, at the following rate:

	s.	d.
Ordinary examination, Bacteriological or Pathological, such as tumour, membrane, or sputum	2	6
Ordinary (qualitative) urine examination	2	6

Any further report will be charged for at a special rate. If a mounted specimen is desired an extra charge of 1s. will be made. If a telegraphic report is required, the cost of the telegram will be charged in addition.

Specimens must be accompanied by the fee and a stamped addressed envelope, in which the report will be sent as soon as possible. Specimens, with, if possible, a short history of the case, must be addressed to "The Manager of the Journal," with "Pathological Department" written in some conspicuous place on the wrapper.

Births.

OLIVE.—Jan. 7th, at Leamington, the wife of E. J. P. Olive, M.D., M.A., F.R.C.S., of twin daughters.

MILLER.—Jan. 23rd, at Iver, Bucks, the wife of John Miller, L.R.C.P., M.R.C.S., of a son.

ROGERS-TILLSTONE.—Jan. 19th, at Ditton Holme, near Maidstone, the wife of J. M. Rogers-Tillstone, M.R.C.S., L.R.C.P.Lond., of a daughter.

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